

INTRODUCTION

The newborn infant is actually a foetus suddenly removed from complete dependence on the maternal organism in an aqueous environment to a relatively independent status in air. Successful transmission from intra to extra-uterine life is dependent on the readjustment of a great number of functions to be accomplished more or less urgently during and following delivery. Amongst these adaptive processes the most dramatic changes are those occurring in the circulatory and respiratory systems. The readjustment processes occurring in these two systems are intimately linked together and are interdependent. At birth the inactive foetal lungs have to take over the function of the placenta as an effective gas exchanger to ascertain survival in the new environment.

Respiratory problems present the major threats to survival of the newborn infant (Driscoll and Smith, 1962). They are overwhelmingly the consequence of infant's failure to complete successfully the respiratory and circulatory adaptation from intra to extra-uterine life.

A great deal of interest and research is centered during recent years on the acid-base and electrolyte homeostasis and on its disturbances in the newborn infant. Duration of labour, mode of delivery, drugs administered during labour, prematurity, low birth weight and toxæmia of pregnancy are the additive factors which may possibly disturb this mechanism in the newborn infants.

The development of satisfactory ultramicro methods has enabled the present research workers to carry out serial determinations of biochemical variables on samples of capillary blood obtained by a heel prick from the neonatal infants.

Several authors have reported the acid-base studies ranging from birth to seven days in the full-term normal and premature infants (Graham, Wilson, Tsao, Mary, Bauman and Brown, 1951; Reardon, Bauman, and Haddad, 1960; Bucci, Scalamandre, Savignoni, and Mendicini, 1965; Jurado-Garcia, Cobos, Napoles, Cazares, Villalba, and Riversa, 1965; Malan, Evans, and Heese, 1965; Yu, Payne, Ifekwunigwe, and Stevens, 1965; Rogner and Frenzel, 1966; Severi, Belloni, Perinotto, and Bergamaschi, 1970; Ray, Ray, Sarkar and Chatterjee, 1972). A few reports are available regarding the serial studies of electrolytes in the full-term normal infants, healthy premature infants and infants delivered by difficult obstetric procedures (Overman, Etheldorf, Bass and Horn, 1951; Rosen, 1961; Acharya, 1962; Yu et al., 1965). However, reports on the simultaneous serial determinations of acid-base and electrolyte parameters in respect of normal, premature, low birth weight infants and infants delivered with maternal toxemia are rare in the literature.

Serial determinations of plasma total protein concentrations in the full-term normal infants were carried out by Graham et al., 1951; Gairdner, Marks, Roscoe and Brettell, 1958; Reardon et al., 1960; Acharya, 1962 and Markarian, Jackson and Bannon, 1966. Similar studies on the premature infants and the infants

delivered by difficult delivery procedures were performed by Yu et al., 1965; Markarian et al., 1966 and Acharya, 1962. Studies of the plasma total protein and its electrophoretic separation on the cord samples of full-term normal infants and their mothers at delivery were reported by Longsworth, Curtis and Pembroke, 1945; Stanier and Thompson, 1954; Jencks, Smith and Durrum, 1956; Oberman, Gregory, Burke, Ross and Rice, 1956; Brown, Mc Gandy, Gillie, Doyle and Albany, 1959; Ramkumar, Singh and Sood, 1964; Hardie, Heese and Kench, 1965; Khalil, Guirgis, Et Khateeb and Samei, 1968 and Ezeilo, 1971. It would be observed from the above work that serial studies on the plasma protein fractions are lacking in the literature.

The chemical composition of the body fluids shows not only species differences, but variations occur as a result of racial, environmental, dietetic and climatic factors. The Indian literature lacks norms for the acid-base and electrolyte data in the newborn infants.

In view of the reasons mentioned in the above paragraphs, the present study was undertaken.

The present work is concerned with the serial determinations of the acid-base and electrolyte parameters and the plasma protein fractions for a period till 96 hours after birth in the full-term normal infants, infants delivered by difficult obstetric procedures, healthy premature infants, healthy low birth weight infants and infants with maternal toxæmia.

The present study can be resolved into the following stages:-

1) Study of serial determinations of acid-base parameters like blood pH, partial pressure of carbon dioxide (pCO_2), base excess (BE), standard bicarbonate (standard HCO_3), actual bicarbonate (actual HCO_3) and carbonic acid (H_2CO_3) in the full-term normal infants.

2) Study of serial determinations of electrolyte parameters like plasma potassium, sodium, chloride, calcium, magnesium, inorganic phosphorus, total protein and blood urea in the full-term normal infants.

3) Serial determinations of electrophoretic separation of plasma protein in the full-term normal infants.

4) Study of the above-mentioned parameters in the other groups of infants.

5) Correlation of the data obtained on account of physiological events occurring during the early neonatal period in the full-term normal infants with that of the other groups.

A complete statistical evaluation is an unattainable perfection during this age period, when the changes in the individual infants show considerably wide variations. However, the trends seen in the mean values of each group of infants appear to be meaningful and are utilized to illustrate the changes.

CLINICAL MATERIAL

The clinical material in the present study consists of the infants delivered at Shree Sayaji General Hospital, Baroda.

Total number of 63 newborn infants (27 males, 36 females) were studied. The record of their sex, weight, maturity score, condition at birth, progress, length of labour, type of delivery and maternal obstetric history were noted. Realising the difficulty in getting the correct information of gestation period from the mothers, the following factors were used for grouping the newborn infants:

(1) Birth weight: The maternity and Child Health Expert Committee of WHO (1961) classified all the term infants with birth weight less than 2500 G. as low birth weight infants and infants born at any time before 37 weeks, as prematures or immatures. (2) Maturity score: It is based on scoring of various physical and neurological criteria suggested by Chikermane, Majmudar and Shah (1969). The maximum score given is 20. Infants with maturity score below 14 are termed as premature infants and those who have 14 or more are termed as low birth weight term infants.

Grouping of the infants was done as follows:-

Full-term normal infants:

Sixteen infants included in this group were delivered by spontaneous vaginal delivery, weighed 2500 G. or more at birth and had maturity score 14 or more.

Infants delivered by difficult obstetric procedures:

Out of 19 infants comprising this group, first 11 were delivered by Caesarean section and the other eight by forceps application for either maternal or foetal indications like prolonged second stage of labour, foetal and/or maternal distress, pelvic contraction and ruptured uterus. Most of them weighed more than 2500 G. at birth and gestational ages were between 37 to 40 weeks. The deliveries by forceps application were carried out under local anaesthesia. Caesarean sections were performed under general anaesthesia. All the mothers undergoing Caesarean section received 5% glucose (or glucose saline) infusions during labour and/or at surgery.

Healthy premature infants:

Ten infants included in this group were delivered by spontaneous vaginal route, weighed less than 2500 G. at birth and had maturity score less than 14.

Healthy low birth weight infants:

Eleven infants studied in this group were delivered by vaginal route, weighed less than 2500 G. at birth and had maturity score 14 or more.

Infants delivered by difficult obstetric procedures with maternal toxæmia:

Three infants in this group were delivered by forceps application with a history of maternal toxæmia as evidenced by oedema, proteinuria and high blood pressure.

Low birth weight infants with maternal toxæmia:

Four infants in this group were delivered by vaginal route, weighed less than 2500 G. at birth, had maturity score 14 or more and had a history of maternal toxæmia.

Clinical data of all the infants and their mothers are recorded in Appendix I.

COLLECTION OF MATERIAL

Arterialized capillary blood was collected from each infant at 0, 2, 4, 8, 12, 18, 24, 48, 72 and 96 hours after birth. All the blood samples were taken by heel puncture after application of a thin film of sterilized paraffin (Pildes, Hart, Warrner, and Cornblath, 1969). The cut was made deep enough to permit a free flow of blood. After wiping away the first drop of blood, a heparinized glass capillary was inserted into the centre of the droplet, to fill it completely. A small piece of iron wire was inserted into the capillary, then the ends were sealed with plasticine, for anaerobic storage of the blood sample. The blood and heparin were mixed by the small iron wire insert, from the external movement of a small magnet. Four capillary samples were collected every time for acid-base measurement and stored in ice water (approx 4° C), till determination was performed within 30 minutes.

Another sample of blood (0.8 to 0.9 ml.) was collected into a clean heparinized Widal tube. The tube was shaken intermitantly during blood collection to give an anticoagulant effect. It was then centrifuged at 3,000 r.p.m. for 10 minutes. Approximately, about 0.3 to 0.4 ml. of plasma was obtained depending upon the haemoconcentration. From this sample of plasma, potassium, sodium, chloride, calcium, magnesium, inorganic phosphorus and total protein were determined. The electrophoretic separations of the plasma proteins were carried

out on all the samples. Urea was determined either on the whole blood or on the packed cells.

Two ml. blood was collected in a heparin container from the antecubital vein of the mother after delivery. The sample was centrifuged and the plasma was used for determination of total protein and electrophoretic separation.

All the plasma samples were stored in the glass stoppered tubes in the refrigerator (2-6° C) till they were analysed after collection of the last sample at 96 hours.

LABORATORY METHODS

During the last decade suitable ultramicro methods have been developed allowing determinations of biochemical values with good accuracy. The laboratory methods used for this study require 10 to 50 μ l of plasma or blood for each parameter. The total volumes were kept as small as possible avoiding high dilutions. Beckman spinco polythelene pipettes (with water repellent surface) were used in this work. At frequent intervals the pipettes were filled with a fresh solution of 1% pepsin in 0.1 N hydrochloric acid and were left overnight to remove any thin invisible coating of protein.

For colorimetry, an Evans Electro selenium Limited, titrator unit with 10 mm light path was used. Minimum fluid volume required to read was 2.0 ml. Standards of low and high concentrations were included in all the batches of the samples. All the techniques were checked from time to time with control sera.

Blood acid-base parameters were determined by means of the micro Astrup method (Siggard Andersen, Engel, Jorensen, and Astrup, 1960). Actual pH was determined at body temperature of 37° C. Other parameters like blood $p\text{CO}_2$, BE, standard bicarbonate and actual bicarbonate were determined according to microequilibration technique described by Siggard Andersen(1963). The carbonic acid is calculated by multiplying $p\text{CO}_2$ with a factor 0.03, which represents a proportionality constant between the dissolved CO_2 and $p\text{CO}_2$.

The plasma potassium and sodium were determined by the E.E.L. flame photometer using external standards. 20 μ l of plasma was used for each determination. The plasma potassium and sodium samples were diluted with 2.0 ml. and 5.0 ml. double distilled water respectively.

The plasma chloride was determined colorimetrically by mercuric nitrate method of Schales and Schales (1941) using 10 μ l of the sample.

The plasma calcium and magnesium were determined by adapting Wilkinson's method (1957) using ethylene diamine tetraacetic acid (E.D.T.A.) to titrate calcium and magnesium ions in the presence of murexide and eriochrome black as indicators. 20 μ l of plasma was used for each determination.

The plasma inorganic phosphorus was determined by the method of Natelson (1961) using 1, 2, 4 aminonaphthol sulfonic acid as a reducing agent. The sample required for estimation was 50 μ l.

Plasma total protein was determined by biuret method (O'Brien, Ibbott and Rodgeron, 1968). Electrophoretic separation of the plasma protein was carried out by E.E.L. electrophoresis apparatus using 10 μ l of the sample and Whatman paper No.1. The strip was stained with lissamine green according to the method of Gorringer (1957). Bands corresponding to protein fractions were eluted and the elute's optical density was read at 630 $m\mu$.

Blood urea was determined by the diacetyl monoxime method using Somogyi filtrate (Nateson, 1961). 50 μ l of the sample was required for the estimation.

FULL-TERM NORMAL INFANTS

ACID-BASE STATUS

The blood pH of 7.4 is slightly to the alkaline side. The maintenance of the pH of the blood is one of the vital interests of the body. There is only a very slight change between the pH of arterial blood (7.43) and venous blood (7.40), though the venous blood has to transport plenty of carbon dioxide. Tissue metabolism continues to pour more and more of carbon dioxide into the blood. The carbon dioxide combines with water to form carbonic acid, the reaction being catalysed by the enzyme carbonic anhydrase present in the red blood cells. Again, due to metabolic reactions, various organic acids like pyruvic acid, lactic acid, acetoacetic acid and inorganic acids like sulphuric, hydrochloric and phosphoric acids are produced and these add H-ions to the blood by their ionization. Blood has, under normal conditions various mechanisms to counteract acidosis and alkalosis, whether respiratory or metabolic. The measurement of pH alone is insufficient for the clinical evaluation of hydrogen ion metabolism (acid-base balance). A serious disarrangement in hydrogen ion metabolism is compatible with a normal absolute concentration of hydrogen ions (normal pH). This paradox is explained by the compensatory mechanisms in the form of the blood buffer systems and the organs (lung, kidney) of hydrogen ion excretion.

RESULTS:

In the present investigation, therefore, all the parameters of acid-base balance viz. blood pH, pCO_2 , BE, Standard HCO_3 ,

actual HCO_3 and H_2CO_3 are studied in 16 full-term normal infants during the first 96 hours of life. The results of their determinations are expressed as mean, standard deviation, range and number in Table 1. Their mean values are illustrated graphically in Fig.1.

pH: The level of blood pH is low with a mean of 7.28 at birth and rises rapidly with the establishment of respiration to lower limits of normal adult value of 7.36 at 12 hours. The pH remains between 7.38 and 7.40 from 18 hours onwards.

pCO₂: The mean pCO₂ value of 45.6 mm.Hg observed at birth is the highest level seen during the 96 hour period. It recedes gradually to 36.3 mm.Hg at 18 hours and ranges from 36.1 to 37 mm.Hg during the remaining period.

BE: The mean base excess at birth is found to be -6.3 mEq/L., which gradually rises to normal level of -1.9 mEq/L. at 24 hours of age and varies from --1.7 to -2.4 mEq/L. during the remaining period.

Standard HCO₃: The standard bicarbonate is a simple estimate of metabolic acidosis or alkalosis. The mean level at birth is 19.5 mEq/L. which shows a steady rise to 22.6 mEq/L. till 48 hours when the mean pH is 7.40. The rise in bicarbonate lags behind the rise in blood pH.

Actual HCO₃: The difference between the actual bicarbonate and standard bicarbonate concentrations indicates a respiratory acidosis or alkalosis. The mean concentration of actual HCO₃ at birth is 20.9 mEq/L. which falls to 19.9 mEq/L. at four

hours. It ranges from 20.4 to 21.6 mEq/L. during the remaining period. The levels of actual bicarbonate are lower than the adult range throughout the observation period of 96 hours.

H₂CO₃: The mean carbonic acid concentration of 1.37 mEq/L. observed at birth is the highest level seen during the 96 hour period. It follows a trend similar to pCO₂.

HCO₃/H₂CO₃: The ratio is 15.33 at birth and rises gradually upto 19.84 at 48 hours when the blood pH is 7.40. Further, a fall is seen with a mean value of 18.93 at 96 hours.

Table 1-A demonstrates the course of changes in the acid-base pattern of the same infant of this group.

At birth the infants are in a combined respiratory and non-respiratory acidosis. Respiratory acidosis is corrected rapidly after two hours. Though the pH of 7.33 at four hours of age is significantly below the average level for adults, the value of pCO₂ (39.5 mm.Hg) indicates that the alveolar ventilation is adequate. The increase in pH is on account of decrease in pCO₂ and the chemical pattern in about 88 per cent of infants is that of non-respiratory acidosis. A gradual correction of the non-respiratory acidosis is evidenced by normal acid-base balance in 56 per cent of the infants at 18 hours. At 24 hours of age, 63 per cent of infants show a normal acid-base pattern, 25 per cent show respiratory basosis and the remaining are in a state of non-respiratory acidosis. Majority of the infants exhibit normal acid-base status during

24 to 72 hours of age as noticed from the mean values of all the parameters. It would be seen from Table 1-A that 12 per cent of the infants show non-respiratory acidosis at 24 and 48 hours. However, the incidence of the non-respiratory acidosis rises to 29 and 33 per cent at 72 and 96 hours respectively. The onset of respiratory basosis occurs at 18 hours, and is maximum (25 per cent) between 24 to 72 hour period.

Representative data from the literature on acid-base status during the early neonatal period are summarized in Table 2.

A serial acid-base study till 96 hours after birth, in full-term infants, was undertaken by Reardon et al. (1960). They noticed a combined acidosis at birth. The non-respiratory acidosis was not associated with a decrease in the bicarbonate content but a decrease in non bicarbonate buffer anions was seen. However, a notable decrease in both bicarbonate and non bicarbonate buffer anions was seen within an hour after birth with normal ventilation. A marked decrease in $p\text{CO}_2$ was observed at 24 hours of age. Respiratory basosis was seen in 18 per cent of the infants from 24 to 72 hours after birth. The trend in the acid-base pattern of the infants in this group and those in the present series is found similar till 24 hours after birth. Thereafter, a divergence in both the groups till 72 hours is seen. The mean values during 24 to 72 hours in present series show a normal acid-base balance while respiratory basosis is found by Reardon and associates (1960).

Malan et al. (1965) carried out a serial acid-base study in the full-term infants for a period of 72 hours after birth. These authors observed initial combined acidosis till 2 hours followed by the correction of the respiratory component at four hours after birth. The residual non-respiratory acidosis was of a minor degree showing a normal acid-base pattern. About 42 per cent of the infants had respiratory basosis at some time, usually after 24 hours. A good correlation between the observations of Malan et al. (1965) and the present series is seen in: (a) correction of combined and respiratory acidosis after two hours, (b) observation of maximum number of infants in non-respiratory acidosis at four hours, (c) improvement in non-respiratory acidosis at 24 and 48 hours, (d) development of respiratory basosis at 24 hours persisting upto 72 hours.

While the differences noted are:

(i) maximum number of infants show normal acid-base status at sixth hour in the study of Malan et al. (1965) and at 24 hours in the present study, (ii) non-respiratory acidosis is virtually absent at 72 hours in the observation of Malan et al. (1965) while its incidence increases from 48 hours onwards till 96 hours in the present series.

Jurado-Garcia et al. (1965) in a cross-sectional study of acid-base parameters in full-term newborn infants reported non-respiratory and respiratory acidosis at birth. These workers observed a much faster gaseous adaptation which attained normal level by 30 minutes with persistence of non-respiratory acidosis. The infants were in a state of fully

compensated respiratory basis at eight hours (pH-7.39, pCO₂ 33.75 mm.Hg) and again entered the non-respiratory acidosis phase from 24 hours onwards. The 96 hour's pattern revealed respiratory basis. The mean values of acid-base parameters in their series indicate that the infants swing from non-respiratory acidosis to respiratory basis during the whole period without attaining normality. The correlation in the findings of this group and the present series is seen upto four hours after birth.

In a longitudinal study in mature newborn infants during the first week of life, Rogner et al. (1966) observed considerable fluctuations around the lower limits of normal pH values for adults. These infants were in a state of non-respiratory acidosis for the whole period showing a gradual compensation from the first to fourth day of life, while a majority of the infants in the present study are in normal acid-base balance from 18 hours after birth.

Koch (1968) studied lung function and acid-base balance in the full-term newborn infants. The author observed respiratory and non-respiratory acidosis at birth with accumulation of lactic and pyruvic acids. Acidosis and concentration of lactic acid increased during the first minutes following birth, but decreased rapidly during the subsequent minutes. Respiratory acidosis was removed within 30 to 60 minutes after birth. At 24 hours after birth, inspite of persistent hypobasemia (standard HCO₃, 20 mEq/L.) a normal pH

value counterbalanced by hyperventilation ($p\text{CO}_2$ 33 mm.Hg) was found. During the further course of the first week, rise in $p\text{CO}_2$ towards lower limits of the normal adult range paralleled by an increase in the bicarbonate concentration was observed.

In a study of birth weight and acid-base equilibrium in the first days of life, Severi et al. (1970) observed a continuance of non-respiratory acidosis till 3 days after birth. The infants were in normal acid-base equilibrium for the remaining period of the study upto 7 days.

A cross-sectional study of acid-base homeostasis of the normal neonates was performed by Ray et al. (1972). They found mixed acidosis in the infants till one hour after birth. The infants were in the state of non-respiratory acidosis upto 24 hours. A pattern of compensated respiratory basosis ($p\text{CO}_2$ 32.81 mm.Hg) seen between two to four days of life changed to non-respiratory acidosis showing an increase in $p\text{CO}_2$ and a decrease in the standard HCO_3 concentrations.

DISCUSSION:

Various mechanisms explaining the acid-base changes during the neonatal period have been suggested by different workers. Wilson, Reardon and Murayama (1948) postulated that anaerobic metabolism, with diminished CO_2 production, is responsible for the low plasma CO_2 content in the newborn infants. The work of Singer, Elkinton, Barker and Clark (1953) indicates the possibility of migration of hydrogen ion from the cell to the extracellular fluid (ECF) during the CO_2 retention. It is possible that the CO_2 retention during parturition induces a similar change in the human infant (Weisbrot, James, Prince, Holaday and Apgar, 1958). Graham et al. (1951) have shown the plasma "R" fraction (which consists largely of organic acids) to be 15 mEq/L. for 24 hours old infants, where as that of the adult is only 10 mEq/L.

Some degree of asphyxia, usually of a brief duration, occurring as a result of the delivery process is a normal finding in all births (James, Weisbrot, Prince, Holaday and Apgar, 1958; James, 1960). The oxygen debt in the infant during the terminal phases of delivery appears to be responsible for the high lactic acid content of the cord blood. The incidence of increased lactic acid concentrations at birth and subsequently for a period of 3 to 5 hours is reported by Acharya (1962). Graham and Wilson (1954) suggest that the changes in pH, pCO_2 and calculated oxygen tension (pO_2), indicate "that a considerable portion of the total stimulus to

respiration is derived from the effect of oxygen lack". These authors have observed the oxygen tension in the blood of many newborn infants to be well below 55 to 60 mm.Hg at birth. This level is low by the adult standards and can be effective in stimulating respiration. Koch and Wendel (1967) found a mean arterial value of pO_2 to be 62.9 mm.Hg even during a span of five hours to six days after birth. Koch (1968) in a further study of lung function and acid-base balance in the newborn infants observed a pO_2 value between 10 to 20 mm.Hg at birth in the umbilical artery. Subsequent levels of pO_2 between 10 to 60 minutes ranged from 50 to 60 mm.Hg. These concentrations of pO_2 are suggestive of the evidence of hypoxia during this early neonatal period.

Ray et al. (1972) have offered a different mechanism for hyperventilation resulting in a fall of pCO_2 level and correction of respiratory acidosis. They postulate that increased concentration of circulating progesterone in the newborn infant during the early postnatal period is responsible for stimulation of the respiratory centre which leads to hyperventilation. This increased progesterone concentration has been thought to be a result of passive hormonal transfer from the mother who shows a similar trend during the last trimester of pregnancy. However, no data are available of progesterone determinations in the neonatal infants during the postnatal hours of life. The presence of anaerobic glycolysis occurring during this period is evident from the work of James et al. (1958), James (1960), Acharya (1962) and Koch (1968).

Ray et al. (1972) have reported the presence of combined acidosis in 80 per cent of their cases at birth for which these workers have not offered an explanation.

In view of the above facts, it would be seen that, it is more reasonable to think that the initial hyperventilation occurring during the postnatal hours is due to stimulation of the respiratory centre in response to hypoxia. Hyperventilation would result in removal of the excess of CO_2 , thus accounting for the correction of respiratory acidosis after 2 hours in the present study.

As a result of hyperventilation the pCO_2 level tends to fall and attain average adult standard by four hours. The hyperventilation continues resulting in a further gradual fall of pCO_2 till 24 hours after birth along with an increase in the standard and actual bicarbonate concentrations. The continuation of hyperventilation from four hours onwards till 24 hours seen in most of the babies could be due to a slow response in the sensibilization of respiratory centre after its stimulation by hypoxia. Such a phenomenon of slow response in the sensibilization of the respiratory centre is observed in the adult man during acclimatization to altitude changes.

The gradual disappearance of non-respiratory acidosis from four to 24 hour period could be on account of diminished lactic acid production due to correction of hypoxia. This is supported by an increase in the base excess concentration seen during the above period. A similar trend of improvement in the

non-respiratory acidosis is observed by most of the workers who have carried out serial studies (Reardon et al., 1960; Malan et al., 1965; Koch, 1968).

The observation of respiratory basosis between 24 to 72 hours in 25 per cent of infants in the present study is due to further continuation of hyperventilation. Similar findings of the onset of respiratory basosis from 24 hours onwards are reported by Reardon et al. (1960); Malan et al. (1965) and Ray et al. (1972). However, none of the above workers except Ray et al. have offered any explanation for this change.

The increasing incidence of non-respiratory acidosis seen after 48 hours onwards is probably due to compensation of respiratory basosis through conservation of fixed acids by the kidney.

TABLE 1. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.40 0.03 16	36.5 2.31 16	-1.8 2.2 16	22.6 1.30 16	21.6 2.88 16	1.09 0.15 16	19.84 0.71 16
72	7.39 0.027 14	37.0 3.10 14	-1.7 2.12 14	22.2 1.50 14	21.4 2.00 14	1.11 0.08 14	19.31 0.88 14
96	7.38 0.03 12	36.1 3.20 12	-2.4 2.88 12	21.6 1.41 12	20.6 2.32 12	1.08 0.10 12	18.93 1.04 12

TABLE 1-A.

3 IN 16 FULL-TERM NORMAL INFANTS

Hours after birth	Non-respiratory acidosis	Normal	Respiratory basosis
0	4 5 8 9 15 12 16 (47)		
2	2 3 5 7 8 9 15 12 13 (56)	6 4	(12)
4	1 2 3 4 5 6 7 8 11 12 13 14 15 16 (88)	9	(6)
8	3 4 5 7 8 9 10 11 12 13 14 15 16 (82)	6 2	(12)
12	3 5 7 8 9 10 11 15 16 (56)	6 2 4 12 13 14	(38)
18	3 7 9 10 11 12 (38)	1 2 4 5 6 13 14 15 16 (56)	8 (6)
24	3 10 (12)	1 2 4 5 6 7 12 13 14 16 (63)	11 8 9 15 (25)
48	3 4 (12)	1 2 5 7 9 14 15 12 16 (57)	11 8 10 13 (25)
72	1 4 7 15 (29)	6 2 5 12 14 10 16 (50)	11 8 9 (21)
96	1 4 11 15 (33)	6 2 9 10 7 12 16 (59)	11 8 (8)

le indicate per cent of infants.

TABLE 2.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE AND CARBONIC ACID CONCENTRATIONS OF FULL-TERM NORMAL INFANTS. (REPRESENTATIVE DATA FROM LITERATURE)

Author (Year) Age	Sample	pH		pCO ₂ mm.Hg		BE mEq/L.		Std.HCO ₃ mEq/L.		Actual HCO ₃ mEq/L.		H ₂ CO ₃ mEq/L.	
		Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	
Graham, Wilson, Tssao, Mary, Bauman & Brown (1951)													
	0-1 hr	7.34 0.047 43	7.20- 7.48	-	-	-	-	-	-	20.0 2.8 43	11.2- 24.5	-	-
	4 hr	7.40 0.042 10	7.33- 7.46	-	-	-	-	-	-	19.0 2.7 10	14.2- 22.5	-	-
	8 hr	7.41 0.040 11	7.35- 7.46	-	-	-	-	-	-	21.0 1.7 11	16.0- 22.4	-	-
	12 hr	7.44 0.043 10	7.36- 7.50	-	-	-	-	-	-	19.0 1.5 10	17.2- 22.1	-	-
	24 hr	7.43 0.049 12	7.36- 7.50	-	-	-	-	-	-	21.0 2.4 12	16.1- 24.9	-	-
Pincus, Gittleman, Saito & Sobel (1956)													
	0-24 hr	7.36 0.07 12	-	35.0 5.0 12	-	-	-	-	-	21.0 3.0	(CO ₂ content)	-	-

TABLE 2. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃	H ₂ CO ₃
Reardon, Baumann & Haddad (1960)	Cord	7.26	53.4	-4.2	-	23.4	-
		7.12- 7.35	28.7- 84.5	-	-	21.1- 27.0	-
	1 hr	7.30	39.1	-7.0	-	18.8	-
		7.21- 7.38	32.7- 57.1	-	-	15.5- 22.4	-
	4 hr	7.33	37.7	-5.7	-	19.5	-
		7.26- 7.42	30.1- 47.5	-	-	15.5- 22.3	-
	24 hr	7.38	33.4	-4.2	-	19.5	-
		7.34- 7.44	21.5- 41.5	-	-	12.9- 17.6	-
	48 hr	7.39	34.1	-3.4	-	20.0	-
		7.31- 7.44	22.4- 42.2	-	-	14.4- 24.7	-
	72 hr	7.38	34.8	-3.6	-	20.4	-
		7.32- 7.45	28.8- 39.0	-	-	17.8- 24.4	-
96 hr	7.39	36.4	-2.3	-	21.4	-	
	7.32- 7.46	29.2- 42.9	-	-	18.2- 24.0	-	
Jurado-Garcia, Cobos, Napolos, Cazares, Villalba & Rivera (1965)	C	7.16	57.37	-	-	19.69	1.78
		0.09	2.83	-	-	2.32	0.28
		23	23	-	-	23	23

TABLE 2. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃	H ₂ CO ₃
30 min	C	7.31 0.08 10	41.67 9.77 10	-	-	20.76 1.53 10	1.35 0.32 10
1 hr	C	7.34 0.04 9	39.37 8.12 9	-	-	20.79 2.22 9	1.20 0.28 9
2 hr	C	7.32 0.06 9	40.67 2.13 9	-	-	20.68 2.95 9	1.26 0.20 9
4 hr	C	7.30 0.08 33	41.35 9.92 33	-	-	19.83 2.16 33	1.30 0.29 33
8 hr	C	7.39 0.03 5	33.75 5.98 5	-	-	19.64 2.59 5	1.05 0.19 5
24 hr	C	7.36 0.12 44	38.55 8.62 44	-	-	19.09 1.71 44	1.21 0.26 44
48 hr	C	7.35 0.14 42	35.53 6.29 42	-	-	18.59 1.74 42	1.10 0.19 42
72 hr	C	7.35 0.13 35	35.94 7.50 35	-	-	19.62 2.42 35	1.12 0.24 35
96 hr	C	7.39 0.13 17	31.84 7.59 17	-	-	18.56 2.89 17	0.98 0.31 17

TABLE 2. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃	H ₂ CO ₃
Malan, Evans & Heese (1965)							
2 hr	0	7.34 0.06 16	41.4 8.8 16	-3.8 2.4 16	21.1 1.8 16	21.3 4.3 16	-
4 hr	0	7.38 0.04 16	36.7 5.3 16	-2.5 1.6 16	22.0 1.3 16	21.4 1.4 16	-
6 hr	0	7.38 0.04 16	36.5 3.6 16	-2.5 1.8 16	21.9 1.6 16	21.4 1.9 16	-
12 hr	0	7.40 0.03 16	36.6 3.3 16	-1.4 1.8 16	22.9 1.7 16	22.1 2.0 16	-
24 hr	0	7.41 0.04 16	34.9 4.0 16	-1.4 1.8 16	22.8 1.6 16	21.7 1.9 16	-
48 hr	0	7.42 0.05 16	34.0 4.5 16	-1.2 1.7 16	23.0 1.6 16	21.3 2.0 16	-
72 hr	0	7.42 0.04 16	35.5 5.4 16	-0.8 2.4 16	23.3 2.1 16	22.2 3.1 16	-
Rogner & Frenzel (1966)							
1 day	0	7.297 0.053	44.4 8.3	-	19.7 1.7	-	-
2 day	0	7.336 0.038	39.4 4.8	-	20.5 1.6	-	-

TABLE 2. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃	H ₂ CO ₃
3 day	C	7.346	39.5	-	20.6	-	-
		0.037	6.4	-	1.6	-	-
		7.353	38.3	-	21.1	-	-
		0.030	4.5	-	1.6	-	-
		7.345	41.9	-	21.4	-	-
5 day	C	0.031	6.0	-	1.3	-	-
		7.350	37.8	-	20.8	-	-
6 day	C	0.035	5.0	-	1.4	-	-
		7.354	37.7	-	20.6	-	-
7 day	C	0.040	6.5	-	1.4	-	-
Sachin & O'Brien (1967)							
1-3 day	C	7.36	33.5	-	-	18.6	-
		0.04	3.5	-	-	2.7	-
4-7 day	C	29	29	-	-	29	-
		7.34	39.6	-	-	20.6	-
8-14 day	C	0.04	6.3	-	-	3.0	-
		29	29	-	-	29	-
Saveri, Belloni, Perinotto & Bergamaschi (1970)	C	7.37	38.5	-	-	22.3	-
		0.03	2.8	-	-	2.4	-
2-3 hr	C	29	29	-	-	29	-
		7.32	35.43	-	18.95	-	-
		0.042	1.89	-6.73	1.53	-	-
		18	18	18	18	-	-

TABLE 2. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃
24 hr	C	7.359	35.84	-4.41	20.74	-	-
		0.019	3.50	2.08	1.36	-	-
		17	17	17	17	-	-
3 day	C	7.360	36.85	-3.64	20.21	-	-
		0.022	4.91	1.58	1.15	-	-
		18	18	18	18	-	-
5 day	C	7.380	39.60	-1.51	22.90	-	-
		0.010	2.61	0.88	0.78	-	-
		18	18	18	18	-	-
7 day	C	7.375	39.12	-1.85	22.84	-	-
		0.010	3.29	1.01	1.00	-	-
		9	9	9	9	-	-
Ray, Ray, Sarkar & Chatterjee (1972)							
0-1 hr	C	7.26	51.23	-5.91	-12.0	19.46	16.5-
		0.02	6.18	2.96	1.56	-	-
		10	10	10	10	-	-
2-24 hr	C	7.33	40.13	-4.5	-12.4	21.93	14.0-
		0.03	5.84	2.9	1.72	-	-
		32	32	32	32	-	-
2-4 day	C	7.39	32.81	-3.2	21.35	-	-
		0.04	3.38	2.71	1.72	-	-
		27	27	27	27	-	-
5-7 day	C	7.34	34.87	-5.75	19.95	-	-
		0.02	2.38	1.32	1.06	-	-
		23	23	23	23	-	-

A = Arterial

C = Capillary

FIGURE 1.

BLOOD pH, pCO_2 , BASE EXCESS, STANDARD BICARBONATE,
ACTUAL BICARBONATE, CARBONIC ACID AND
BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF
FULL-TERM NORMAL INFANTS (•—•).

The scale is reduced after 18 hours.

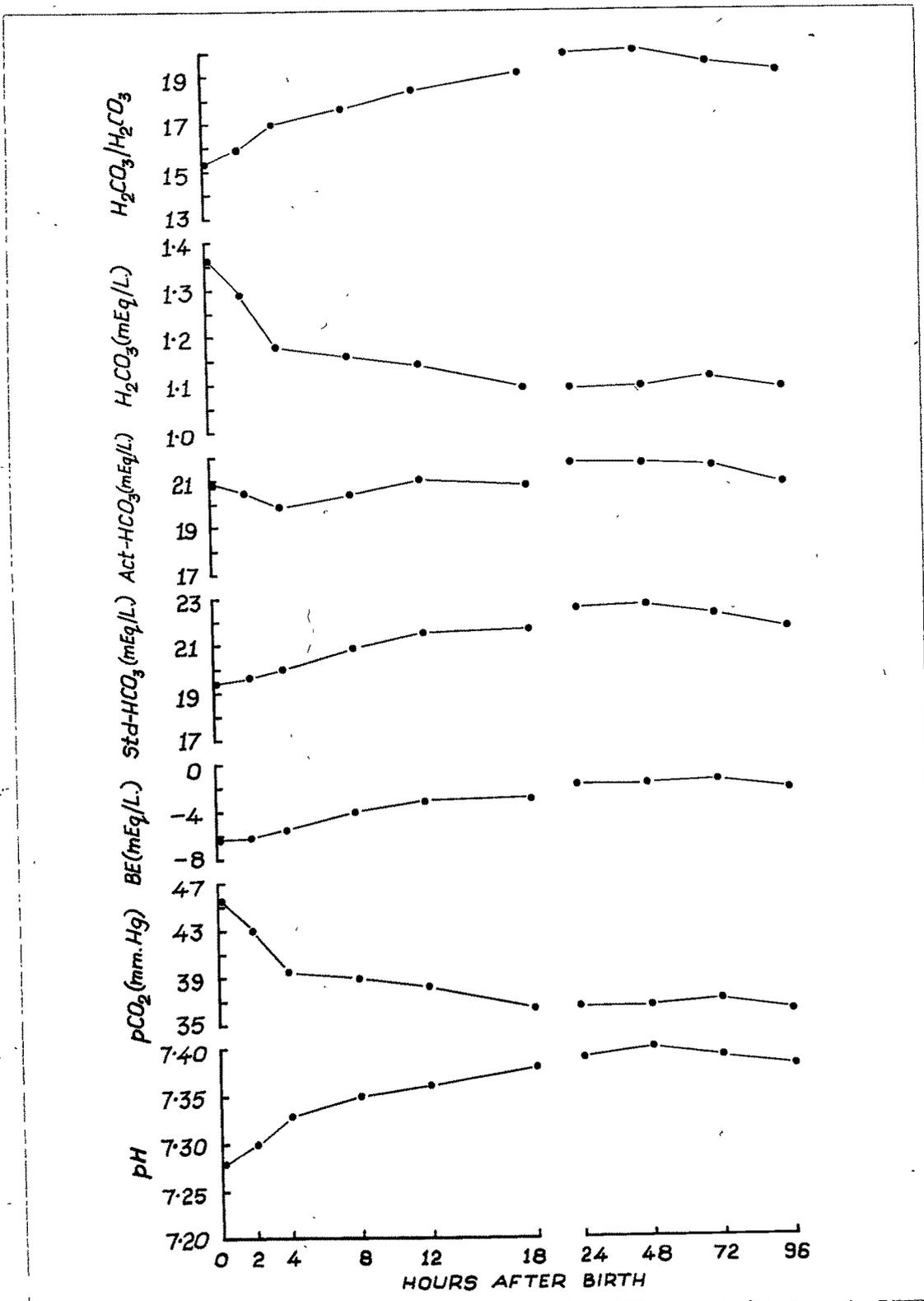


Figure 1.

PLASMA ELECTROLYTE STATUS

The importance of electrolytes in the living being cannot be ignored. Homeostasis of electrolyte balance is one of the many problems before the neonate after birth. Variance of electrolyte concentration could be on account of more than one factors. Potassium is the main cation of the intracellular fluid. Maintenance of its concentration in the extracellular fluid is necessary for muscular activity, particularly that of cardiac muscle. Sodium and chloride are the largest cation components of extracellular fluid. Sodium in association with chloride and bicarbonate helps in the regulation of acid-base equilibrium. Maintenance of osmotic pressure of the body is another important role of sodium. It also helps in the preservation of normal irritability of muscle and permeability of the cells. Calcium and phosphorus are important constituents of bone and tooth. Calcium helps in coagulation, enzyme reactions, muscle contraction and integrity of the intercellular cement substances, while phosphorus is essential for high energy bonds, synthesis of phospholipids, cell membranes and nervous tissue. Magnesium is the second most intracellular cation. It plays an important role as a metallic activator of enzyme system. Like calcium, it has an influence on the excitability of the component of neuromuscular apparatus. Proteins are the primary constituents of the body. The proteins of blood plasma help in controlling the distribution of fluids in the body and provide antibodies to combat disease. Proteins

also help in the oxygen transport, buffering mechanism and regulation of chemical processes in the form of enzymes and hormones. Urea is the chief end product of protein metabolism. It forms the largest fraction of nonprotein nitrogenous substance in the blood and contributes about 80 per cent of the total urinary nitrogen.

RESULTS:

In the present study, the parameters of electrolyte balance, i.e. plasma potassium, sodium, chloride, calcium, magnesium, inorganic phosphorus, total protein and blood urea are studied in 16 full-term normal infants during the first 96 hours of life. Table 3 and 6 show the results of their determinations expressed as mean, standard deviation, range and number. The behaviour of the mean concentrations of these parameters is shown in Fig.2.

Plasma Potassium: The mean plasma potassium concentration at birth is 5.2 mEq/L. It is slightly higher than the adult value. The concentration falls to 5.0 mEq/L. at four hours and remains steady between 12 to 24 hours at 5.1 mEq/L. Plasma potassium concentration is found in the normal adult range after 48 hours.

Plasma Sodium: The mean plasma sodium concentrations vary within narrow limits of 141.4 to 143.2 mEq/L. with the highest value at birth. A fall in the sodium level is seen between 24 to 72 hours.

Plasma Chloride: The mean plasma chloride concentration is within the normal adult range of 95 to 105 mEq/L. The mean value shows a rising tendency, reaching the highest level of 104.2 mEq/L. at the age of 18 hours. This is 5 per cent higher than the lowest value of 98.3 mEq/L. at birth.

Plasma Calcium: The mean plasma calcium concentration of 10.7 mg./100 ml. seen at birth is highest in the series. It falls upto the age of 24 hours reaching minimum value of 9.7 mg./100 ml. The fall is 9.3 per cent. Thereafter, plasma calcium concentration is more or less constant and the mean values are within the normal adult range throughout the period of study.

Plasma Magnesium: Like calcium, highest concentration of plasma magnesium is also seen at birth. The value is 30 per cent higher than the lowest value of 1.34 mg./100 ml. at 12 hours. Plasma magnesium values do not show much variation afterwards. The mean plasma magnesium concentrations are less than the lower limits of normal adult range after four hours.

Plasma Inorganic Phosphorus: The mean inorganic phosphorus concentration is 4.99 mg./100 ml. at birth. It increases upto the age of 48 hours reaching maximum value of 6.3 mg./100 ml. The rise is 26 per cent. The mean inorganic phosphorus concentrations are higher than the normal adult range throughout the period of observation.

Plasma Total Protein: The mean total protein levels vary within narrow limits. The highest value, 7.1 g./100 ml. at

birth, being 9 per cent higher than the lowest value, 6.52 g./100 ml. at 48 hours. Plasma protein concentration during the period of study remains within the normal adult range and shows a gradual fall till 48 hours after birth.

Blood Urea: Blood urea level at birth is 15.8 mg./100 ml. It increases upto 48 hours after birth attaining maximum level of 26.7 mg./100 ml. The rise is about 70 per cent. The mean blood urea concentrations are within the normal range and show a trend similar to phosphorus.

Representative data from the literature on electrolyte status during the early neonatal period are summarized in Table 4 and 7.

Graham et al. (1951) found normal plasma chloride levels during the first day of life. In a serial study of plasma sodium, potassium and chloride, Overman et al. (1951) observed values within the normal range from birth to 48 hours, except an increased plasma chloride concentration at 24 hours. Rosen (1961) studied plasma sodium, potassium, chloride and blood urea concentrations during the first three days of life. Plasma potassium showed a gradual fall from 4.94 to 4.50 mEq/L. Plasma sodium was on the lower limits of the adult range at birth. Plasma chloride and blood urea showed a gradual rise.

Acharya (1962) carried out serial electrolyte estimations including plasma sodium, potassium, chloride, calcium, magnesium, inorganic phosphorus, protein and blood urea for a period of 48 hours after birth. The author has reported much

higher values of plasma potassium at birth. It showed a considerable fall at one hour, followed by a moderate rise at three hours. Thereafter, a slow fall for rest of the 48 hour period was seen. The plasma potassium concentration did not reach the normal adult level even by 48 hours. The mean concentration of sodium showed some fluctuations in two to seven hour period, after which a rise throughout the rest of the 48 hour period was seen. The plasma chloride concentration showed a variation during the first nine hours, followed by a slow rise upto 24 hours and finally adjusted at the cord blood level. The mean concentration of plasma calcium fell remarkably at the fifth hour after birth and continued to fall upto 24 hours. It showed a slight rise at the end of 36 hours. The mean concentration of plasma magnesium in the cord blood was found to be 0.9 mg./100 ml. Plasma magnesium concentration after a slight rise at three hours remained below the lower limits of normal range during the period of study. The plasma inorganic phosphorus concentration showed a significant rise upto three hours, after which an abrupt fall with only small variations occurred. The mean concentration of plasma protein showed a significant rise at the end of one hour. A considerable variation was seen in the two to 18 hour period after which there was a consistent rise in the plasma protein concentration. The mean blood urea concentration showed a marked fall at two hours with a steady rise upto 18 hours.

Anast (1964) found the magnesium concentration of cord blood to be lower than magnesium levels during the first week of life. Harvey et al. (1970) observed plasma calcium and magnesium concentrations lower than the adult range during the seven day period of study, except the finding of normal calcium level on the third day.

Jukarainen (1971) studied plasma calcium, magnesium and inorganic phosphorus concentrations in 73 full birth weight infants for 13 times during the first five days of life. Plasma calcium concentration fell from the initial level 9.6 to 7.8 mg./100 ml. at 44 hours. This was a fall of 18.5 per cent. The mean magnesium concentration at birth was 2.07 mg./100 ml. The lowest value of 2.01 mg./100 ml. at 82 hours, was 7 per cent lower than the highest, 2.17 mg./100 ml. at the age of 20 hours. During the five days, the mean magnesium concentration in this group showed no significant change with age. The level of inorganic phosphorus at birth was 6.7 mg./100 ml. increased upto the age of 60 hours, reaching maximum value of 7.9 mg./100 ml. The rise was 18.5 per cent.

DISCUSSION:

Available reports on the plasma potassium concentration at birth are contradictory. Overman et al. (1951), Pincus et al. (1956) and Solomkin and Tauber (1959) have observed plasma potassium values slightly higher than the adult range, while Widdowson and McCance (1956), Oliver et al. (1961) and Acharya (1962) have reported much higher values. Widdowson and McCance (1956) noticed an elevated potassium concentration in the foetal and neonatal pigs. They suggested that foetal cells may be less able than adult cells to take in potassium against a high concentration gradient. Acharya (1962) reported much higher plasma potassium concentrations than the normal adult values throughout the period of 48 hours after birth. The author was of the opinion that during the early postnatal period several factors are operating so as to effect changes in the plasma potassium concentration. The increase in plasma potassium at birth could be attributed to deficiency of adrenal cortex hormones, tissue destruction, glycogen breakdown and acidosis. Anaesthesia is known to cause a transient acidosis which results in lowering of pH and an increase in extracellular potassium. The mothers of the full-term normal infants in the present series were not given any anaesthesia. The absence of anaesthesia during labour may account for lower concentrations of mean plasma potassium at birth in the present series as compared to the above workers. However, the mean values of plasma potassium are slightly higher than the normal adult

range. This increase in plasma potassium is due to hypoxia leading to a milder degree of acidosis at birth (pH 7.28). Slight fall in plasma potassium observed at fourth hour may be due to removal of respiratory component of acidosis. The subsequent somewhat higher mean values of plasma potassium are due to the persistence of non-respiratory acidosis.

The data on serum or plasma sodium concentrations of the normal newborn infants reported by most of the workers are within the normal adult range. Acharya (1962) observed a tendency towards a fall in plasma sodium concentrations in a considerable number of infants during the first 12 hours of life. He attributed this change to the deficiency of mineralocorticoids. The author reported a subsequent rise in the plasma sodium concentration resulting from hydropenia occurring during the early days of life. The mean plasma sodium concentrations in the present investigation are within the normal adult range and show no significant changes to comment upon.

The rise seen in the mean concentrations of plasma chloride in the postnatal period by Overman et al. (1951), Acharya (1962) and present series is on account of hydropenia as suggested by Smith (1959).

Bakwin (1937, 1939) studied the serum calcium level during the early neonatal period and found a gradual fall associated with a rise in the inorganic phosphate concentration during the first day of life. The author attributed this fall to a

transient hypofunction of parathyroids. His concept was based on the following facts: (1) whatever maternal hormone might have been directly or indirectly affecting the foetus is suddenly withdrawn at birth; (2) most infants show an early postnatal decrease in serum calcium concentrations; (3) reduction in the urinary excretion of phosphate (similar to that occurring in hypoparathyroidism) is demonstrable in the newborn infants; and (4) many infants have been shown to develop still more striking hypocalcaemia if phosphorus is ingested, much as do parathyroid-deficient older subjects. The observations of Todd et al. (1939) are in agreement with those of Bakwin (1937). Danzer (1939) and Acharya (1962) did not find a negative correlation between the postnatal fall in plasma calcium and phosphorus after birth. Acharya (1962) attributed the positive correlation of simultaneous fall in the plasma calcium and inorganic phosphorus concentrations to their utilization for mineralization of the bone. Bergman (1971) postulated that an increase in the ionized plasma calcium level stimulates the release of thyrocalcitonin which eventually results in a fall in the plasma calcium concentration. This author further stated that mineralization of the bone and temporary hypofunction of parathyroid may also be additive factors in producing this change. Jukarainen (1971) suggested that the interaction paralleling between plasma calcium and phosphorus may possibly reflect skeletal mineralization.

The trend of the fall observed in the plasma calcium

concentrations during 24 hour period in the present series is accompanied by a concomitant rise in the plasma inorganic phosphorus level. These findings are in agreement with Bakwin's (1937) hypothesis of transient hypoparathyroidism occurring during the postnatal hours. However, the suggestions of Bergman (1971) regarding the role of bone mineralization and release of thyrocalcitonin in the regulation of calcium homeostasis cannot be ignored. An abrupt fall in the plasma inorganic phosphorus occurring between eight to 12 hour period may be on account of bone mineralization resulting in the formation of calcium phosphate complex as suggested by Acharya (1962) and Bergman (1971). Subsequently plasma inorganic phosphorus level shows a tendency to rise till 48 hours. This can be on account of tissue breakdown coupled with deficient renal function (McCance and Widdowson, 1954) as evidenced by a similar rise observed in the blood urea concentrations.

Anast (1964) supported his observation of cord blood magnesium concentration to be lower than those during the first week of life, on the basis of role of placenta in magnesium metabolism in utero. While the findings of Bajpai, Sugden, Ramos and Stern (1966) are opposite. These workers have offered no explanation for their observations. Jukarainen (1971) is of the opinion that hypoparathyroidism induced by high phosphorus concentration can account for the inverse relationship seen between magnesium and phosphorus

concentrations. Similar negative correlation between magnesium and phosphorus concentrations is observed in the present series during the first 12 hours of life and can be explained on the same basis. Subsequent behaviour of magnesium concentration does not show much variation to comment upon.

The total plasma protein concentrations show a gradual fall during the first 48 hours of life. Similar fall in serum/plasma protein concentrations has been reported by Graham et al. (1951), Overman et al. (1951), Reardon et al. (1960) and Hardie et al. (1965). This fall in protein concentration may be due to fluid shift from vascular space to the tissues on account of an increased capillary permeability resulting from hypoxia at birth.

Urea is the chief end product of protein metabolism. Late rise observed in the blood urea concentration upto 48 hours could be explained on the basis of deficient renal clearance and tissue breakdown. The improvement in renal function and/or tissue breakdown would account for the subsequent fall in the blood urea and plasma inorganic phosphorus concentrations seen after 48 hours.

TABLE 3.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS
OF FULL-TERM NORMAL INFANTS. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
0	5.2 0.70 15	4.3- 7.0	143.2 6.75 15	134- 160	98.3 5.62 15	89- 105	10.74 0.84 14	8.8- 11.9	1.75 0.59 12	0.9- 2.8	4.99 1.05 14	3.2- 6.6	15.8 4.28 15	12- 27
2	5.2 0.56 16	4.5- 6.7	142.6 7.11 16	132- 156	101.1 9.62 16	89- 123	10.18 0.90 15	9.1- 11.9	1.74 0.61 15	0.5- 2.9	5.11 1.38 15	3.3- 9.1	16.8 5.66 16	11- 32
4	5.0 0.55 16	4.1- 6.2	142.6 7.51 16	132- 158	102.4 7.61 16	89- 114	10.52 1.11 15	8.8- 12.5	1.59 0.42 15	0.9- 2.3	5.56 1.55 15	3.9- 9.7	18.0 5.53 16	11- 32
8	5.0 0.46 16	4.2- 6.0	142.7 7.23 16	136- 158	103.4 7.24 16	89- 115	10.06 1.09 16	8.6- 12.1	1.44 0.39 16	0.9- 2.0	5.33 1.61 16	2.8- 9.7	19.2 4.24 16	11- 28
12	5.1 0.43 16	4.1- 6.1	141.5 4.35 16	134- 150	103.8 6.35 16	96- 118	9.96 1.05 16	8.0- 11.9	1.34 0.21 16	1.1- 2.8	5.29 0.88 16	4.1- 7.0	20.9 5.72 16	15- 35
18	5.1 0.30 16	4.7- 6.0	142.9 3.25 16	138- 150	104.2 5.43 16	96- 114	9.82 0.95 16	8.0- 11.0	1.45 0.39 16	0.7- 2.1	5.57 0.85 16	4.2- 7.5	23.9 4.43 16	16- 32
24	5.1 0.55 16	4.4- 6.1	143.1 5.66 16	132- 152	102.8 5.89 16	91- 114	9.70 0.80 16	8.6- 10.9	1.35 0.44 16	0.5- 2.1	6.06 1.23 16	4.2- 8.8	24.7 6.65 16	15- 34

TABLE 3. (CONTINUED)

Hours after birth	K	Na	Cl	Ca	Mg.	Phosphorus	Urea
48	5.22 0.84 16	141.7 5.61 16	101.7 5.27 16	9.81 0.78 16	1.40 0.43 16	6.30 1.37 16	26.7 11.12 16
72	4.68 0.56 14	141.4 4.93 14	102.3 7.46 14	10.07 0.84 14	1.34 0.43 14	5.80 1.15 14	22.4 14.4 14
96	4.87 0.72 11	142.2 3.93 12	103.6 6.00 12	9.98 0.99 11	1.36 0.53 10	5.88 1.07 12	21.7 8.16 12

TABLE 4.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF FULL-TERM NORMAL INFANTS. (REPRESENTATIVE DATA FROM LITERATURE)

Author (Year) Age	K mEq/L.		Na, mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg. mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	SD+		SD+		SD+		SD+		SD+		SD+		SD+	
	No.		No.		No.		No.		No.		No.		No.	
Bakwin (1937)														
Cord	-	-	-	-	-	-	11.0	-	-	-	-	-	-	-
							0.92							
							300							
0-2 hr	-	-	-	-	-	-	10.29	-	-	-	5.7	-	-	-
							-				-			
							7				7			
3-5 hr	-	-	-	-	-	-	9.76	-	-	-	6.14	-	-	-
							0.66				0.82			
							31				30			
6-10 hr	-	-	-	-	-	-	10.36	-	-	-	6.84	-	-	-
							0.5				0.62			
							23				24			
24 hr	-	-	-	-	-	-	9.57	-	-	-	-	-	-	-
							-				-			
							7				-			
Denzer, Reiner & Weiner (1939)														
Cord	-	-	-	-	-	-	11.53	9.00-	-	-	6.06	4.0-	-	-
							-	13.12			-	8.6		
							91				41			
1-2 day	-	-	-	-	-	-	10.48	8.75-	-	-	5.71	4.4-	-	-
							-	11.88			-	7.1		
							21				14			

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
Data from 22 authors summarized by Todd, Chuinard & Wood (1939)							
Cord	-	-	-	40.97 8.30- 14.4 832	-	4.3 1.9- 13.3 645	-
Todd et al. (1939)							
Cord	-	-	-	11.27 7.3- 16.9 121	-	5.55 4.2- 8.0 121	-
1-3 day	-	-	-	9.93 7.2- 12.3 630	-	6.08 3.7- 8.6 548	-
McCance & Widdowson (1947)							
Cord	-	-	-	-	-	-	18.9 8.0- 28.8 12
3-3½ day	-	-	-	-	-	-	29.1 18.5- 39.6 12

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
Graham et al. (1951)							
0-1 hr	-	-	104.5 2.16 43	-	-	-	-
4 hr	-	-	103.9 2.5 10	-	-	-	-
8 hr	-	-	106.1 2.2 11	-	-	-	-
12 hr	-	-	104.5 1.4 10	-	-	-	-
24 hr	-	-	105.3 3.2 12	-	-	-	-
Overman, Etheldorf, Bass & Horn (1951)							
Cord	5.0 1.01 19	134.0 4.2 19	109.0 6.3 19	-	-	-	-
Birth	4.1 0.49 17	134.0 2.8 17	105.0 4.9 17	-	-	-	-

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na.	Cl	Ca	Mg.	Phosphorus	Urea
Gittleman, Pincus, Schmerzler & Saito (1956)							
1 day	-	-	-	9.2 0.6 824	-	5.7 0.7 824	-
						4.0- 7.4	-
Pincus et al. (1956)							
0-24 hr	5.4 0.7 12	135.0 5.0 12	102.0 6.0 12	-	-	-	15.0 7.0 12
Widdowson & McCance (1956)							
Cord	8.0 - 5	138.0 134- 146 5	110.0 107- 111 5	-	-	-	-
	4.8- 12.9						
Solomkin & Tauber (1959)							
Cord	5.1 - 15	139.0 124- 150 15	104.7 95- 112 18	-	-	-	-
	4.7- 5.8						
Usher (1959)							
12-60 hr	6.01 - 13	-	-	-	-	-	-

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
Rosen (1961)							
1 day	4.94 0.80 29	134.8 5.2 30	94.3 4.5 30	-	-	-	24.5 9.0 31
2 day	4.55 0.49 29	136.3 4.3 29	95.8 6.2 30	-	-	-	28.6 9.5 30
3 day	4.50 0.67 24	136.1 4.0 25	99.8 5.1 27	-	-	-	26.8 12.6 27
Oliver, Demis & Bates (1961)							
Cord	9.1 2.0 19	139.0 8.0 19	107.0 4.0 11	-	-	-	-
1 hr	8.0 1.8 24	134.0 5.0 24	109.0 4.0 20	-	-	-	-
Acharya (1962)							
0 hr	7.79 2.00 14	146.78 8.1 14	103.28 4.63 14	9.34 - 5	0.93 - 3	5.65 1.28 14	29.37 7.3 13
1 hr	6.78 0.71 13	145.38 8.23 13	102.53 5.68 13	8.95 - 4	1.23 - 3	6.05 1.5 14	29.71 10.4 12

TABLE 4. (CONTINUED)

Author (Year) Age)	K	Na	Cl	Ca	Mg	Phosphorus	Urea
2 hr	6.65 0.83 13	145.5 8.42 14	121- 155 81- 106	-	-	6.29 1.42 14	24.3 8.1 13
3 hr	7.0 0.94 13	142.0 10.4 14	109- 159 83- 114	8.92 - 5	1.4 - 3	6.35 1.47 14	25.7 9.3 13
5 hr	6.84 0.73 11	143.57 7.2 14	124- 156 90- 111	8.38 - 5	1.23 - 3	6.1 1.57 14	27.45 6.6 13
7 hr	6.4 0.64 12	143.49 5.8 14	130- 153 94- 117	8.32 - 5	1.16 - 3	5.91 1.48 14	27.6 9.7 13
9 hr	6.71 0.79 13	144.93 4.1 14	130- 159 5683- 115	-	-	5.73 1.53 14	28.52 12.2 13
11 hr	6.6 0.79 13	146.2 5.6 14	132- 159 82- 114	8.22 - 5	1.2 - 3	5.68 1.51 14	32.29 11.8 13
18 hr	6.33 0.78 13	145.5 7.3 14	132- 159 87- 114	7.84 - 5	1.06 - 3	5.74 1.64 14	33.45 11.9 13
24 hr	6.19 0.73 14	146.42 6.5 14	131- 156 97- 119	7.7 - 5	1.16 - 3	5.71 1.61 14	31.84 12.9 13
36 hr	6.03 0.63 14	147.78 6.9 14	134- 160 92- 114	8.02 - 5	1.23 - 3	5.93 1.6 14	32.53 14.0 13
48 hr	5.92 0.8 14	148.7- 4.3 14	139- 162 93- 112	7.94 - 5	1.33 - 3	5.84 1.57 14	30.76 12.9 13

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
Anast (1964)							
Cord	-	-	-	-	1.89 0.26 41	1.43- 2.45	-
1 day	-	-	-	-	1.94 0.26 56	1.40- 2.90	-
2 day	-	-	-	-	1.87 0.27 56	1.36- 2.90	-
3 day	-	-	-	-	1.95 0.24 55	1.41- 2.59	-
4 day	-	-	-	-	1.91 0.27 49	1.36- 2.62	-
5 day	-	-	-	-	2.01 0.28 22	1.49- 2.73	-
Harvey, Cooper & Stevens (1970)							
2 day	-	-	-	7.5 - 9	1.2 - 9	0.9- 1.8	-
3 day	-	-	-	9.3 - 9	1.4 - 9	0.9- 1.7	-

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
4 day	-	-	-	8.6 7.2- 10.2	1.3 1.1- 1.6	-	-
5 day	-	-	-	8.3 2.4- 12.6	1.10 0.42- 1.86	-	-
6 day	-	-	-	8.6 6.4- 10.2	1.6 1.1- 1.71	-	-
7 day	-	-	-	8.9 6.8- 11.4	1.36 1.1- 1.6	-	-
Jukarainen (1971)				mEq/l.	mEq/l.		
0-2 hr	-	-	-	4.8 0.5 53	1.73 0.31 42	6.7 2.1 48	-
2-8 hr	-	-	-	4.6 0.5 34	1.72 0.25 24	6.7 2.1 27	-
8-16 hr	-	-	-	4.3 0.6 65	1.73 0.34 56	7.2 2.0 47	-
16-24 hr	-	-	-	4.1 0.6 60	1.81 0.31 49	7.1 2.2 52	-
24-32 hr	-	-	-	4.1 0.5 68	1.71 0.27 62	7.0 1.8 56	-

TABLE 4. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
32-40 hr	-	-	-	4.0 0.5 60	1.70 0.26 53	7.5 1.8 53	-
40-48 hr	-	-	-	3.9 0.6 64	1.70 0.25 55	7.7 1.6 52	-
48-56 hr	-	-	-	4.0 0.6 65	1.71 0.28 58	7.9 1.9 55	-
56-64 hr	-	-	-	4.0 0.5 63	1.72 0.27 55	7.9 2.3 52	-
64-76 hr	-	-	-	3.9 0.6 64	1.69 0.28 55	7.8 2.1 53	-
76-88 hr	-	-	-	4.1 0.6 51	1.68 0.28 44	7.6 1.8 45	-
88-100 hr	-	-	-	4.2 0.5 54	1.70 0.31 48	7.5 1.8 47	-
100-128 hr	-	-	-	4.2 0.6 39	1.76 0.29 34	7.7 1.9 30	-

FIGURE 2.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM,
MAGNESIUM, INORGANIC PHOSPHORUS, TOTAL PROTEIN AND
BLOOD UREA CONCENTRATIONS (MEAN) OF FULL-TERM NORMAL
INFANTS (•———•).

The scale is reduced after 18 hours.

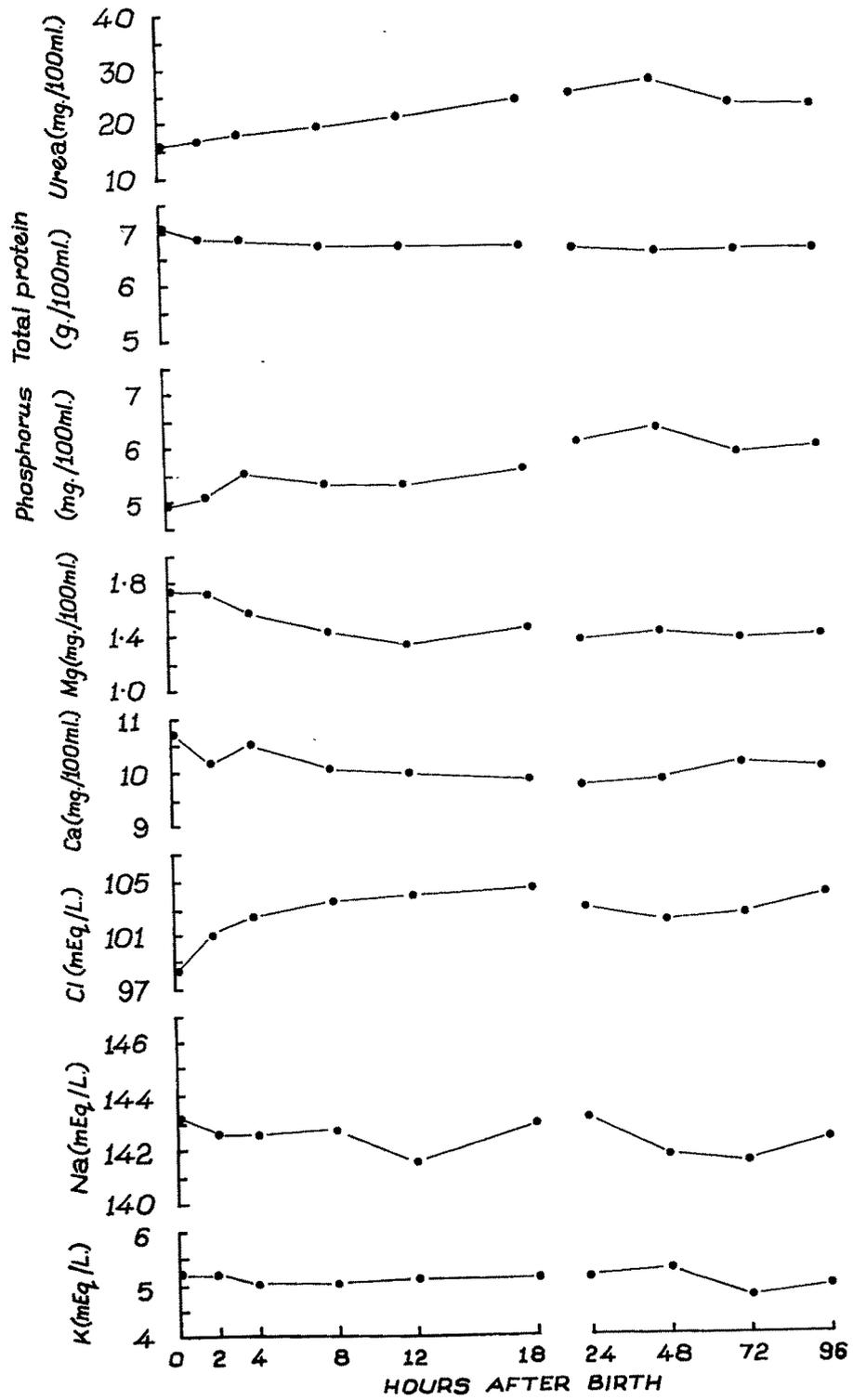


Figure 2.

PLASMA PROTEIN FRACTIONS

The electrophoretic separation is based on the principle that plasma proteins in solution at pH value above and below their isoelectric points migrate in an electric field towards the pole bearing a charge opposite to that of the protein. Albumin fraction normally contributes about 80 per cent of the effective osmotic pressure of plasma. Alpha globulin helps in transporting copper as ceruloplasmin while beta globulin serves to transport iron in the blood in the form of a complex siderophilin or transferrin. Alpha and beta globulins also help in transporting lipids in the form of lipoproteins. Defence mechanism of the body is looked after by gamma-globulins.

RESULTS:

In the present work, electrophoretic separation of plasma proteins is studied in 35 full-term normal infants during the first 96 hours of life and their mothers at delivery. The full-term infants delivered by spontaneous vaginal route and difficult obstetric procedures (forceps application or Caesarean section) are grouped as 'full-term normal infants' for total protein and electrophoretic studies because their mean total plasma protein concentrations did not show any significant differences (Table 5, Fig.4). The results of their determinations are expressed as mean, standard deviation, range and number in Table 6.

Plasma Albumin: The mean plasma albumin concentration of 4.39 g./100 ml. seen at birth is highest in the series. It falls upto eight hours and rises attaining the original value at 18 hours of age. Further, there is a gradual fall to the lowest value of 4.08 g./100 ml. at 72 hours. The fall is 7 per cent. Plasma albumin concentration throughout the period of study is within the normal adult range, while maternal value is much lower than the normal.

Plasma Alpha₁ and Alpha₂ Globulin: The mean concentrations in case of alpha₁ and alpha₂ globulins during the period of study do not show much variation except a rise seen in the alpha₂ level after 24 hours.

Plasma Beta Globulin: The concentration of 0.58 g./100 ml. at birth shows a gradual fall upto 18 hours and a steady rise from 24 hours of age onwards. It attains the original concentration at 96 hours. There is a difference of 18 per cent between the highest concentration of 0.6 g./100 ml. at 2 hours and lowest concentration of 0.49 g./100 ml. at 18 hours.

Plasma Gamma Globulin: The mean concentration of 1.58 g./100 ml. of plasma gamma globulin observed at birth is highest in the series. It falls upto the age of 96 hours reaching the minimum value of 1.27 g./100 ml. The fall is 20 per cent.

Concentrations of the infants' globulin fractions are much lower than those of the mother.

Representative data from the literature on serum/plasma protein electrophoretic patterns during the early neonatal

period are summarized in Table 7.

Many reports are available on the plasma protein electrophoretic separation of the cord sample and mother's blood at delivery, but a serial study on the same infants during the early neonatal period is not found in the literature. The concentration of plasma total protein in the mother's sample is seen to be higher than that in the cord sample in all these studies, while in the present study plasma protein concentrations in the mothers at delivery are lower than those of heel capillary samples of the full-term infants at birth. The electrophoretic patterns of the infants at birth and the mothers at delivery in the present series are in agreement with those of Stanier and Thompson, 1954; Jencks et al., 1956; Ramkumar et al., 1964 and Khalil et al., 1968. This refers to the higher albumin and lower globulin fractions of the infants as compared to those of the mother. The tendency of fall in the total protein, albumin and gamma globulin concentrations seen by Hardie et al. (1965) is also observed in the present series. A higher gamma globulin fraction in the cord plasma as compared to that of the mother was found by Longsworth et al. (1945), Oberman et al. (1956), Brown et al. (1959) and Ezeilo (1971).

DISCUSSION:

It has been shown that amino-acids cross the placenta readily. Early in gestation, the foetal liver is able to synthesize all the plasma protein fractions, except gamma globulin (Dancis, Braverman and Lind, 1957). A detailed study of transfer of plasma proteins from the mother to foetus at term was carried out by Gitlin and Colleagues (1964) using human plasma proteins labelled with ^{131}I . They studied 133 women and their infants and used proteins of various sizes such as acid glycoprotein, albumin, transferrin, gamma G-globulin (Ig G), fibrinogen, gamma M-globulin (Ig M). The concentration in the foetus relative to that in the mother decreased from unity or more for Ig G through acid glycoprotein, albumin, transferrin, Ig M to fibrinogen. In some instances little or no labelled Ig M, transferrin, fibrinogen or even albumin were present. Levels of these substances were low, or they were absent in the amniotic fluid, and there was no evidence of any transfer through the amniotic fluid. The concentration of plasma albumin in the present group is higher than that reported by most of the workers in the literature. It may be due to an adequate synthesis of albumin in the foetal liver. The 7 per cent fall observed in the albumin concentration coupled with simultaneous disappearance of the total protein into tissue spaces may be due to an increased capillary permeability.

Dancis et al. (1957) have demonstrated that the foetal liver of three to four months gestation and placenta at term

are capable to synthesize alpha and beta globulins but not gamma globulin. These findings rule out the possibility of passive transfer of alpha and beta globulins through the placenta. Thus it could be seen that the observation of lower concentrations of alpha and beta globulins in the neonate at birth as compared to that of the mother (present group) may be on account of either deficient synthesis by the foetal liver and/or placenta at term. The fall in beta globulin to an extent of 18 per cent between 18 to 24 hour period may be due to fluid shift as mentioned above. A considerable rise in the α_2 and beta globulins (34 and 20 per cent respectively) is observed from 24 hours onwards till 96 hours in the present series. The author does not have an appropriate explanation to offer for this change.

The foetus has no measurable gamma globulin early in the gestational life. Their appearance and increase begin at 20 to 24 weeks (Rimington and Bickford, 1947). The foetus has a slightly higher concentration of serum gamma globulins than the mother by the time of term birth (Orlandini, Sass-Kortsak and Ebbs, 1955). Dancis and Shafran (1958) have shown that gamma globulin fractions are transferred intact across the guinea-pig placenta and that before birth this is their only source. This means gamma globulins are passively transferred through placenta to the foetus and attain the peak value by term. The gamma globulin concentration of the infant at birth is comparable to that of the mother in the present group.

This supports the above concept of passive transport. The marked decrease of 20 per cent in the gamma globulin concentration is indicative of an increased catabolism of the circulating antibodies and contributes a major part of fall in the total plasma protein concentration.

TABLE 5.
 MEAN PLASMA TOTAL PROTEIN (g./100 ml.) OF FULL-TERM NORMAL INFANTS AND INFANTS DELIVERED
 BY DIFFICULT OBSTETRIC PROCEDURES. (PRESENT SERIES)

	Hours after birth									
	0	2	4	8	12	18	24	48	72	96
Full-term normal infants:	7.10	6.88	6.85	6.73	6.71	6.67	6.62	6.52	6.53	6.54
Infants delivered by difficult obstetric procedures	7.02	7.08	6.83	6.74	6.67	6.72	6.70	6.62	6.60	6.62

TABLE 6.

PLASMA TOTAL PROTEIN AND PROTEIN FRACTIONS (g./100 ml.) OF FULL-TERM NORMAL INFANTS AND THEIR MOTHERS AT DELIVERY. (PRESENT SERIES)

Hours after birth	T. Protein	Globulin													
		Albumin			Alpha ₁			Alpha ₂			Beta			Gamma.	
		Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range	Mean SD± No.	Range		
Mothers at delivery	6.51 0.67 24	5.10- 7.50	2.91 (44.7) 0.64 24	1.82- 4.20	0.31 (4.76) 0.10 24	0.15- 0.54	0.66 (10.14) 0.20 24	0.27- 1.14	0.99 (15.21) 0.27 24	0.56- 1.65	1.64 (25.19) 0.41 24	1.08- 2.15			
0	7.06 0.73 28	5.05- 8.35	4.39 (63.2) 0.58 26	2.85- 5.20	0.17 (2.5) 0.06 26	0.06- 0.30	0.35 (5.0) 0.12 26	0.16- 0.59	0.58 (8.1) 0.18 26	0.23- 0.90	1.58 (21.2) 0.33 26	0.71- 2.08			
2	7.00 0.70 34	5.10- 8.40	4.32 (62.0) 0.62 35	2.95- 5.85	0.20 (2.8) 0.08 35	0.06- 0.36	0.37 (5.2) 0.11 35	0.17- 0.69	0.60 (8.5) 0.22 35	0.30- 1.11	1.51 (21.5) 0.35 35	0.74- 2.19			
4	6.84 0.64 35	5.10- 8.20	4.24 (62.0) 0.67 35	2.83- 6.05	0.20 (2.9) 0.1 35	0.08- 0.42	0.37 (5.4) 0.12 35	0.18- 0.77	0.56 (8.3) 0.18 35	0.28- 1.05	1.48 (21.4) 0.31 35	0.85- 2.06			
8	6.73 0.70 35	5.10- 8.20	4.22 (62.7) 0.62 35	2.85- 5.40	0.18 (2.7) 0.08 35	0.05- 0.37	0.36 (5.3) 0.13 35	0.16- 0.72	0.55 (8.2) 0.20 35	0.34- 1.20	1.42 (21.1) 0.33 35	0.81- 2.01			
12	6.69 0.76 35	4.70- 8.40	4.24 (63.8) 0.59 35	3.20- 5.70	0.20 (2.9) 0.09 35	0.06- 0.52	0.36 (5.1) 0.12 35	0.14- 0.66	0.53 (7.9) 0.19 35	0.24- 1.02	1.36 (20.3) 0.36 35	0.57- 1.96			

TABLE 6. (CONTINUED)

Hours after birth	T. Protein	Albumin	Globulin							
			Alpha ₁	Alpha ₂	Beta	Gamma				
18	6.70 0.74 35	4.38 (65.4) 0.62 34	0.20 (2.8) 0.1 34	0.03- 0.39	0.34 (5.0) 0.34 34	0.16- 0.67	0.49 (7.4) 0.19 34	0.22- 0.95	1.29 (19.4) 0.31 34	0.60- 1.95
24	6.66 0.67 35	4.30 (65.1) 0.75 34	0.19 (2.7) 0.08 34	0.03- 0.34	0.32 (4.8) 0.12 34	0.13- 0.57	0.49 (7.4) 0.18 34	0.26- 1.06	1.35 (20.0) 0.31 34	0.78- 1.93
48	6.57 0.72 34	4.14 (63.1) 0.58 34	0.20 (3.1) 0.08 34	0.07- 0.44	0.35 (5.3) 0.15 34	0.18- 0.75	0.53 (8.0) 0.16 34	0.34- 1.02	1.34 (20.5) 0.32 34	0.86- 2.09
72	6.57 0.69 32	4.08 (62.2) 0.62 32	0.22 (3.2) 0.09 32	0.09- 0.45	0.38 (5.8) 0.11 32	0.19- 0.62	0.56 (8.5) 0.17 32	0.25- 0.92	1.33 (20.3) 0.31 32	0.78- 2.03
96	6.59 0.70 30	4.10 (61.9) 0.67 30	0.21 (3.3) 0.09 30	0.04- 0.51	0.43 (6.5) 0.16 30	0.23- 0.79	0.59 (9.0) 0.18 30	0.28- 0.92	1.27 (19.3) 0.27 30	0.77- 1.75

Figures in the parenthesis indicate percentage of total protein.

TABLE 7. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma
			Alpha ₁	Alpha ₂	Beta	
12 hr	5.98 5.32- 7.03 10	-	-	-	-	-
24 hr	5.71 4.97- 6.34 12	-	-	-	-	-
Overman et al. (1951): Birth	6.2 5.1- 7.5 17	-	-	-	-	-
24 hr	6.1 4.2- 6.7 22	-	-	-	-	-
48 hr	5.7 4.4- 6.7 20	-	-	-	-	-
Stanier & Thompson (1954): Mothers at del- ivery	5.61 4.44- 7.06	2.45 1.93- 3.22	0.98	0.73- 1.33	0.75 0.42- 1.07	1.42 0.89- 1.97
Cord	5.31 4.80- 6.48 14	3.26 2.80- 4.19 14	0.73 0.73 0.98 14	0.37- 0.98	0.26 0.11- 0.43 14	1.03 0.66- 1.52 14

TABLE 7. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma	
			Alpha ₁	Alpha ₂	Beta		
Jencks, Smith & Durrum (1956):							
Mothers at del- ivery	6.60	3.80	0.33	0.73	0.90	0.84	
Cord	5.70	3.94	0.14	0.41	0.44	0.77	
Oberman, Gregory, Burke, Ross & Rice (1956):							
Mothers at del- ivery	6.52 0.16 26	4.69- 7.93 26	2.58 3.39 26	0.47 0.29- 0.65 26	0.63 0.48- 1.12 26	1.42 0.90- 3.26 26	1.21 0.81- 1.58 26
Cord	6.16 0.08 26	5.32- 6.98 26	3.09 3.95 26	0.40 0.23- 0.68 26	0.68 0.38- 0.90 26	0.73 0.38- 1.63 26	1.27 0.82- 1.67 26
3-6 day	6.25 0.10 19	5.44- 6.86 19	3.08 3.76 19	0.35 0.20- 0.51 19	0.60 0.36- 0.78 19	1.06 0.52- 2.83 19	1.15 0.84- 1.54 19
Saito, Gittleman, Pincus & Sobel (1956):							
0-24 hr	5.8 0.57 14	3.31 0.49 14	0.28 0.07 14	0.50 0.12 14	0.51 0.15 14	1.19 - 14	-

TABLE 7. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma
			Alpha ₁	Alpha ₂	Beta	
24 hr	5.59	-	-	-	-	-
48 hr	5.51	-	-	-	-	-
72 hr	5.59	-	-	-	-	-
96 hr	5.43	-	-	-	-	-
Acharya (1962):						
Cord	6.13 0.67 10	-	-	-	-	-
1 hr	6.77 0.73 9	-	-	-	-	-
2 hr	6.52 0.56 6	-	-	-	-	-
3 hr	6.73 0.6 9	-	-	-	-	-
5 hr	6.61 0.63 9	-	-	-	-	-
7 hr	6.6 0.7 10	-	-	-	-	-
9 hr	6.84 0.56 6	-	-	-	-	-

TABLE 7. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma
			Alpha ₁	Alpha ₂	Beta	
11 hr	6.6 0.66 9	-	-	-	-	-
18 hr	6.63 0.68 9	-	-	-	-	-
24 hr	6.78 0.72 10	-	-	-	-	-
36 hr	6.93 0.73 10	-	-	-	-	-
48 hr	7.17 0.76 10	-	-	-	-	-
Udani & Panvalkar (1963):						
Cord	5.87 - 14	3.36 - 14	0.33 - 14	0.50 - 14	0.58 - 14	1.10 - 14
Ramkumar, Singh & Sood (1964):						
Mothers at del- ivery	6.17 0.36 75	2.66 0.42 75	0.32 0.08 75	0.68 0.20 75	0.96 0.21 75	1.55 0.42 75

TABLE 7. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma				
			Alpha ₁	Alpha ₂	Beta					
6 hr	6.24 0.17 12	-	-	-	-	-				
25 hr	6.25 0.14 15	-	-	-	-	-				
48 hr	6.40 0.18 12	-	-	-	-	-				
Khalil, Guirgis, Khateeb & Samei (1968):										
Mothers at del- ivery	6.55 0.36 19	3.66 0.57 19	0.31 0.16 19	0.06- 0.58	0.72 0.20 19	0.34- 1.05	0.79 0.21 19	0.43- 1.19	1.07 0.38 19	0.49- 1.76
Cord	5.90 0.60 19	3.94 0.76 19	0.21 0.13 19	0.03- 0.42	0.38 0.24 19	0.03- 0.74	0.35 0.20 19	0.03- 0.80	1.02 0.41 19	0.24- 1.69
Ezeilo (1971):										
Mothers at del- ivery	5.95 0.19 5	2.38 - 5	0.56 - 5	-	0.97 - 5	-	1.94 - 5	0.91 - 5	-	-
Cord	5.30 0.07 75	2.88 - 75	0.10 - 75	-	0.42 - 75	-	0.67 - 75	1.19 - 75	-	-

INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES

ACID-BASE STATUS

RESULTS:

As mentioned earlier, this group includes infants delivered by instrumental aid, such as Caesarean section or forceps application for either maternal or foetal complications. The acid-base parameters are studied in 19 infants during the first 96 hours of life. The results of these determinations are expressed as mean, standard deviation, range and number in Table 8. Fig.3 illustrates the changes graphically.

pH: The pH value at birth in this group is significantly lower ($t = 2.38$, $P < 0.05$) than that of the normal. Two and four hour values show an insignificant difference, thereafter the values attain the lower limit of normal adult range at 12 hours and show similarity with those of the normal group.

pCO₂: The mean pCO₂ values are lower throughout the period of observation in this group as compared to those of the normal.

BE: The mean base excess concentration is -8.3 mEq/L. at birth which gradually rises to -2.4 mEq/L. after 24 hours. The values do not show significant differences in comparison to those of the normal group.

Standard HCO₃: The standard bicarbonate values are significantly lower than those of the normal group at zero hour ($t = 2.4$, $P < 0.02$) and two hours after birth ($t = 2.56$, $P < 0.02$). Thereafter, the values in both the groups do not show much difference.

Actual HCO_3^- : The mean concentration of 18.8 mEq/L. at birth is lower by 2.0 mEq/L. than that of the normal group, but two hour sample is rather much lower than that of the normal group ($t = 2.50$, $P < 0.02$). Then the differences between the normal and the present group are not of any significance.

H_2CO_3 and $\text{HCO}_3^-/\text{H}_2\text{CO}_3$: The mean concentrations of these parameters follow a pattern similar to those of the normal group.

Table 8-A demonstrates the course of changes in the acid-base pattern of the same infant of this group.

The infants are in the state of combined respiratory and non-respiratory acidosis at birth. Respiratory acidosis is corrected after four hours. At the age of eight hours, 79 per cent of infants show non-respiratory acidosis with a pH of 7.34. A quicker recovery from the non-respiratory acidosis accompanied by a concomitant increase in the incidence of respiratory basosis is seen during the 12 to 24 hour period. The swing from non-respiratory acidosis to respiratory basosis results in a delayed normalization of the acid-base balance. At 72 hours of age, 56 per cent of infants show normal acid-base balance, 33 per cent exhibit respiratory basosis and the remaining are in the non-respiratory acidosis. The onset of respiratory basosis occurs at four hours and is about 30 per cent between 18 to 96 hours. It can be observed from these results that: (a) the rate of correction of non-respiratory acidosis is faster in this group than the normal, (b) the rate of establishment of normal acid-base balance is slower in this group than the normal, (c) the

duration and percentage of infants exhibiting respiratory basosis in this group are higher than those of the normal group.

Representative data from the literature on acid-base status during the early neonatal period are summarized in Table 9.

Jurado-Garcia et al. (1965) have done a cross-sectional study of acid-base parameters for a period of 96 hours after birth in the infants delivered by difficult obstetric procedures. The trend of changes in the acid-base status observed by these authors in this group was similar to that seen in the normal infants, with slight higher magnitude.

DISCUSSION:

The infants delivered by difficult obstetric procedures undergo a more traumatic birth. This leads to a greater degree of hypoxia as compared to the normal infants which is seen from the persistence of the respiratory acidosis for somewhat longer duration in this group. In order to combat the initial high degree of hypoxia, the respiratory centre is stimulated more which results in a greater degree of hyperventilation in a larger percentage of infants. This accounts for an increased incidence of respiratory basosis in this group. The longer duration of the respiratory basosis could be due to a relatively more poor sensibilization of the respiratory centre after an initial higher stimulation. This also accounts for the slow attainment of normal acid-base status in this group.

TABLE 8.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE, CARBONIC ACID AND BICARBONATE/CARBONIC ACID CONCENTRATIONS OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES. (PRESENT SERIES)

Hours after birth	pH		pCO ₂ mm. Hg		BE mEq/L.		Std. HCO ₃ mEq/L. Plasma		Actual HCO ₃ mEq/L. Plasma		H ₂ CO ₃ mEq/L. Plasma		HCO ₃ /H ₂ CO ₃	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
0	7.24 0.07 13	7.11- 7.30	45.7 7.15 13	40.0- 55.0	-8.3 3.90 13	-15.6 -1.0	17.5 2.39 13	14.5- 21.3	18.8 3.45 13	13.1- 21.0	1.37 0.22 13	1.00- 1.65	13.70 2.11 13	10.0- 16.0
2	7.27 0.063 19	7.09- 7.35	40.0 5.68 19	31.5- 50.0	-7.7 4.0 19	-18.2 -1.5	17.8 2.84 19	12.2- 22.7	17.9 3.85 19	13.0- 26.5	1.20 0.17 19	0.95- 1.44	14.79 2.02 19	9.9- 17.6
4	7.30 0.046 19	7.20- 7.36	39.7 6.72 19	30.0- 51.0	-6.8 2.82 19	-11.7 -1.8	19.0 2.16 19	15.2- 22.5	19.1 2.47 19	14.0- 26.0	1.19 0.18 19	0.90- 1.53	16.07 1.48 19	12.8- 18.2
8	7.34 0.046 19	7.29- 7.39	38.4 2.88 19	32.0- 43.0	-5.0 2.0 19	-9.8 -1.0	20.3 1.37 19	16.7- 23.0	19.9 1.91 19	15.0- 23.0	1.15 0.09 19	0.96- 1.29	17.28 1.22 19	15.5- 19.2
12	7.36 0.033 19	7.31- 7.40	37.8 4.04 19	30.0- 47.0	-3.9 1.48 19	-7.5 -1.0	21.2 0.76 19	18.3- 23.2	20.6 2.41 19	17.5- 24.5	1.14 0.10 19	0.93- 1.41	18.18 0.88 19	16.2- 20.0
18	7.38 0.041 19	7.32- 7.45	36.6 7.42 19	26.0- 48.0	-2.9 1.83 19	-6.0 +0.5	21.8 1.62 19	19.4- 24.0	20.9 3.38 19	16.5- 25.5	1.10 0.16 19	0.78- 1.44	19.20 1.42 19	16.6- 22.4
24	7.39 0.033 19	7.23- 7.44	36.1 6.42 19	27.0- 56.0	-2.7 1.93 19	-6.6 +0.7	21.9 0.84 19	19.4- 24.3	20.7 3.97 19	16.5- 24.5	1.09 0.29 19	0.81- 1.68	19.20 1.66 19	13.4- 21.8

TABLE 8. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.39 0.041 18	35.1 2.92 18	-2.4 3.43 18	22.0 0.77 18	20.8 3.72 18	1.05 0.20 18	19.82 1.43 18
72	7.34- 7.50 18	16.0- 42.0 18	-13.8 +1.5 18	15.0- 25.0 18	9.5- 24.0 18	0.48- 1.23 18	17.6- 24.9 18
96	7.40 0.034 18	35.6 4.53 18	-1.8 1.90 18	22.5 0.96 18	21.3 2.21 18	1.07 0.16 18	19.93 1.70 18
	7.37- 7.47 18	20.0- 41.0 18	-10.8 +0.5 18	16.3- 24.5 18	11.5- 24.0 18	0.49- 1.24 18	18.3- 24.7 18

TABLE 8-A.

COURSE OF ACID-BASE STATUS IN 19 INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES

Hours after birth	Combined acidosis	Respiratory acidosis	Non-respiratory acidosis	Normal	Respiratory basosis
0	2 5 12 14 (31)	7 (7)	3 4 6 8 11 13 15 17 (62)		
2	12 (10)	7 19 (10)	1 2 3 4 5 6 8 9 10 11 13 15 16 17 18 (80)		
4	12 (5)	7 16 19 (16)	1 2 4 5 6 8 9 10 11 13 14 15 17 18 (74)		3 (5)
8			1 2 5 6 8 9 10 11 12 14 15 16 17 18 19 (79)	7 4 13 (16)	3 (5)
12		5 17 (10)	1 10 6 11 12 13 15 16 13 (48)	7 8 4 9 (26)	2 3 14 (16)
18		12 17 (10)	5 14 16 18 (21)	7 4 9 10 11 13 19 (37)	1 2 3 8 15 (32)
24	12 (5)		6 8 5 18 (21)	7 9 10 11 16 17 19 (37)	1 2 3 4 13 14 15 (37)
48			1 5 6 8 9 (33) 16 (33)	7 15 11 12 13 17 18 19 (45)	2 4 10 14 (22)
72			9 16 (11)	4 5 6 7 8 11 13 15 18 19 (56)	1 2 10 12 14 17 (33)
96			9 10 16 (17)	4 5 7 11 12 13 14 17 18 19 (55)	1 2 8 15 (28)

Figures shown in the circle indicate per cent of infants.

TABLE 9.

BLOOD pH, $p\text{CO}_2$, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE AND CARBONIC ACID CONCENTRATIONS OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES.
(REPRESENTATIVE DATA FROM LITERATURE)

Author (Year) Age	Sample	pH		$p\text{CO}_2$ mm. Hg.		BE mEq/L.		Std. HCO_3 mEq/L.		Actual HCO_3 mEq/L.		H_2CO_3 mEq/L.	
		Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
		SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.
Jurado-Garcia et. al. (1965)	Birth	7.13	-	62.72	-	-	-	-	-	19.91	-	1.94	-
		0.13		4.48						2.67		0.48	
		16		16						16		16	
	30 min	7.29	-	43.12	-	-	-	-	-	19.81	-	1.34	-
		0.17		13.72						1.37		0.43	
		5		5						5		5	
	1 hr	7.32	-	37.90	-	-	-	-	-	19.26	-	1.18	-
		0.08		7.19						2.05		0.22	
	5		5						5		5		
2 hr	7.31	-	37.90	-	-	-	-	-	18.83	-	1.19	-	
	0.02		2.11						1.44		0.07		
	5		5						5		5		
4 hr	7.29	-	44.75	-	-	-	-	-	21.17	-	1.41	-	
	0.01		10.07						3.00		0.29		
	16		16						16		16		
8 hr	7.38	-	33.00	-	-	-	-	-	18.83	-	1.03	-	
	0.04		7.67						2.18		0.24		
	5		5						5		5		
24 hr	7.31	-	35.13	-	-	-	-	-	18.90	-	1.09	-	
	0.14		9.23						3.37		0.28		
	21		21						21		21		

TABLE 9. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃
48 hr	C	7.33	32.69	-	-	17.98	1.02
		0.06	8.51	-	-	1.87	0.27
		20	20	-	-	20	20
72 hr	C	7.35	35.08	-	-	18.64	1.08
		0.12	9.06	-	-	3.31	0.32
		17	17	-	-	17	17
96 hr	C	7.39	32.46	-	-	19.10	0.90
		0.08	12.33	-	-	2.55	0.18
		12	12	-	-	12	12

C = Capillary

FIGURE 3.

BLOOD pH, $p\text{CO}_2$, BASE EXCESS, STANDARD BICARBONATE,
ACTUAL BICARBONATE, CARBONIC ACID AND
BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF
FULL-TERM NORMAL INFANTS (• ——— •) AND INFANTS
DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES (▼ — — — ▼).

The scale is reduced after 18 hours.

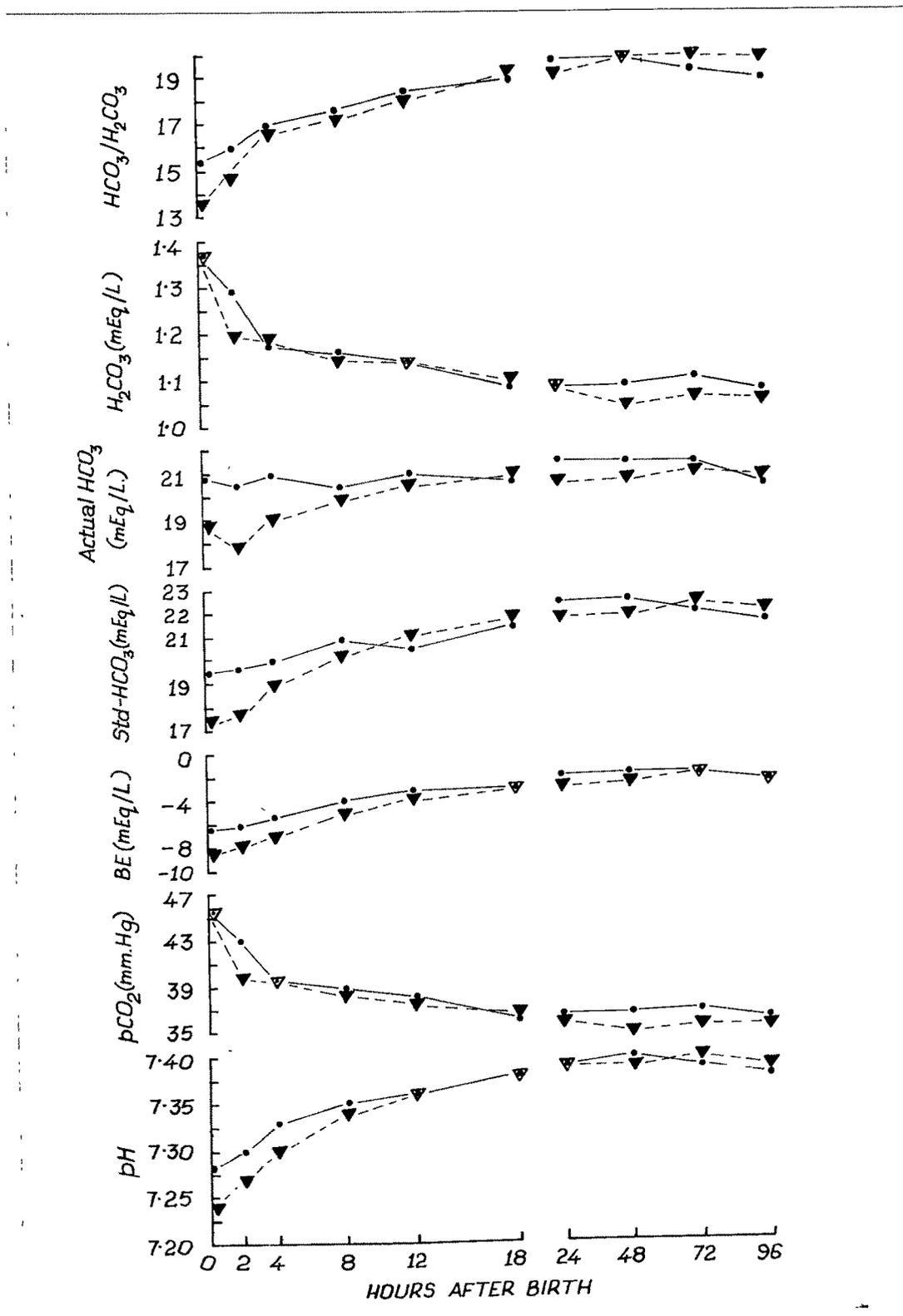


Figure 3.

PLASMA ELECTROLYTE STATUS

RESULTS:

The electrolyte parameters are studied in 19 infants during the first 96 hours after birth. The results of the determinations are expressed as mean, standard deviation, range and number in Table 10. The behaviour of the mean concentrations of these parameters is shown graphically in Fig.4.

Plasma Potassium: The mean concentration of plasma potassium is higher than that of the normal infants during the 24 hour period after birth. The differences are significant at zero, two, four and eight hours after birth ($P < 0.02$, < 0.01 , < 0.001 and < 0.01 respectively). A gradual fall amounting to 20 per cent in the plasma potassium concentration is observed during the whole period though the values are higher than the normal adult range till 48 hours of age.

Plasma Sodium: The mean concentration of plasma sodium is lower till 48 hours of age as compared to that seen in the normal infants.

Plasma Chloride: The plasma chloride concentrations are same at birth in the normal infants and in the infants of the present group. Thereafter, lower concentrations are found throughout the period of observation in the present group as compared to those in the normal. The differences in the concentrations are significant at eight, 12, 18 and 24 hours of age ($P < 0.05$, < 0.01 , < 0.01 and < 0.02 respectively).

Plasma Calcium: The mean concentration of plasma calcium in the present series is lower than that of the normal group during the 96 hour period. Significant difference in the concentration is seen at 48 hours after birth ($t = 2.22$, $P < 0.05$). However, the pattern is similar to that of the normal group.

Plasma Magnesium: The behaviour of the mean concentration of plasma magnesium is similar to that in the normal group with a 30 per cent fall at 24 hours in the basal value of 1.75 mg./100 ml. The concentrations are less than the lower limits of the normal adult after four hours of age.

Plasma Inorganic Phosphorus: The mean concentration of inorganic phosphorus is higher than that of the normal group throughout the period of observation except at four hours. At 72 hours of age, the difference in the concentration is significant ($t = 2.71$, $P < 0.02$) and a rise of 24 per cent is seen.

Blood Urea: The blood urea concentration in this group shows a considerable rise during the whole period as compared to that in the normal group. Significant differences in the concentrations are found at eight, 12, 18, 24 and 72 hours after birth ($P < 0.05$, < 0.001 , < 0.05 , < 0.05 and < 0.02 respectively). The blood urea value of 39.7 mg./100 ml. at 72 hours shows a rise of 82 per cent as compared to the concentration of 21.8 mg./100 ml. at birth.

Representative data from the literature on electrolyte status during the early neonatal period in the infants.

delivered by difficult obstetric procedures are summarized in Table 11.

Rosen (1961) studied electrolyte patterns in the Bantu babies born by Caesarean section during the first week of life. The concentration of plasma potassium was higher than the adult range on the first day and remained within the normal limits afterwards. Plasma sodium was on the lower limits of the normal range except on the third and fifth day. A gradual increase in the concentration of plasma chloride from 96.3 to 104.5 mEq/L. was seen between second to sixth day. Blood urea in this group was significantly higher ($P < 0.001$) than that of the normal on each of the first three days of life. Further, the mean blood urea continued to rise until it reached a high level of 44.1 mg./100 ml. on the fourth day and receded afterwards.

A serial electrolyte study till 48 hours after birth in the infants delivered by difficult obstetric procedures was carried out by Acharya (1962). The author found cord plasma potassium values lower than those of the normal infants. The concentration of plasma potassium was maintained at a slightly higher level than that of the normal group from seven hours of age. A lower concentration of plasma sodium as compared to that in the normal neonates was found throughout the whole period of observation. The plasma chloride concentration did not show much variation from that of the normal. The mean concentration of plasma calcium followed a pattern similar to the normal

group without significant difference. The mean concentrations were higher than those of the normal group though the values were below the range of the normal adult. The mean concentrations of plasma inorganic phosphorus were also higher than those of the normal infants, but the values were higher than the normal adult range. Blood urea concentration in this group showed a late rise between 24 to 48 hours after birth.

DISCUSSION:

The electrolyte changes observed in the infants of difficult delivery group are well marked. The most striking changes are low plasma sodium, low plasma chloride, low plasma calcium, a high potassium, an elevated inorganic phosphorus and a late rise in the blood urea concentrations as compared to those of the full-term normal infants. The results in the present series are comparable to those of Acharya (1962), except the concentrations of plasma potassium, chloride and calcium.

McCance and Widdowson (1954a) studied 10 full-term postnatal infants who were delivered by forceps application and concluded from their results that events connected with a long and difficult labour seem to give rise to a syndrome in the infant which is characterized by (i) a relatively large excretion of inorganic phosphate during and after birth, (ii) an increased destruction of body protein during the first 48 hours of life, (iii) a low urine volume, reduced glomerular filtration rate and a poor urea clearance, (iv) a reduction in the contribution of potassium and chloride to the total osmolarity of the urine. The authors explained these changes on the basis of increased tissue catabolism or breakdown.

Birth involves a certain degree of trauma, and it is not surprising that the homeostatic mechanisms maintaining the internal equilibrium may be affected by the degree of trauma inflicted during birth. The infants delivered by difficult obstetric procedures undergo a more traumatic birth. This

would result in a greater degree of hypoxia and tissue breakdown. The higher concentration of plasma potassium observed during the first eight hours would be on account of the above factors.

The observations of Klien (1951) during the neonatal period are interesting. The author studied the effect of an exogenously administered adrenocorticotrophic hormone (A.C.T.H.) on 12 newborn infants for the periods of one to 10 days. This was accompanied by an expected increase in the excretion of 17-Ketosteroids and urinary nitrogen. The urinary excretion of potassium showed moderate increase on the day of injection. Sodium excretion was always increased a day after an adequate dose of A.C.T.H. administration. When desoxycorticosterone acetate (D.C.A.) was administered to these newborns, they reacted like adults, showing decreased excretion of sodium. This would suggest that the kidney of the newborn is capable of responding normally to adrenocortical hormone. The altered response seen in the neonatal infants after A.C.T.H. administration could be on account of secretion of weak sodium retainers (Klein, 1951). Lanman (1953) reported a similar response to A.C.T.H. during the early neonatal period. Acharya (1962) reported a higher urinary excretion of sodium on the first day after birth as compared to the urinary excretion on the second day in the infants of difficult delivery group. The author suggested that an increased excretion of urinary sodium on the first day could

be due to a rapid effect of the endogenous A.C.T.H. on account of a more traumatic birth. An increased urinary excretion of sodium would favour low plasma sodium concentration. Similar mechanism of an altered response to A.C.T.H. could be responsible for the low concentrations of plasma sodium and chloride observed in the present group as compared to those of the normal group.

The plasma calcium concentration is lower in this group as compared to that of the normal group. This change is significant at 48 hours. Could this be a reciprocal change to balance an increased concentration of inorganic phosphate seen in these infants after 24 hours? The behaviour of plasma magnesium in this group does not show much variation as compared to the normal group.

The rise in the plasma inorganic phosphorus concentration could be due to CO_2 retention (Cordier and Piery, 1955), to liberation from the cell when glycogen is broken down to glucose, or to tissue breakdown. A comparatively higher degree of hypoxia at birth in the infants of the present group results in an increased acidosis as evidenced by the changes in pH, BE and standard HCO_3 concentrations. This increased acidosis could account for the initial high plasma inorganic phosphorus concentrations. The late rise in the inorganic phosphorus concentration seen in the present group cannot be due to CO_2 retention as the pH, pCO_2 and H_2CO_3 values are normal during the period of rise. Glycogenolysis alone cannot

account for such an increase in the inorganic phosphate concentration. Tissue breakdown occurring during the second day seems to be the reasonable explanation for this change.

The rise in the blood urea concentration showing significant differences after eight hours reflect an increased tissue breakdown. The magnitude and duration of tissue breakdown are more in these infants as compared to those of the normal group. This is supported by parallel changes in the plasma inorganic phosphorus concentrations.

TABLE 10.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.
0	5.85 0.70 13	4.5- 7.0	142.0 6.05 13	136- 158	98.3 6.3 13	89- 108	10.50 0.74 12	9.4- 11.7	1.75 0.45 11	1.2- 2.7	5.82 1.38 12	3.5- 8.8	21.8 12.28 13	9- 54
2	5.70 0.53 19	5.0- 6.9	142.4 4.14 19	136- 150	99.0 5.51 19	87- 112	10.34 1.04 19	8.8- 11.8	1.65 0.50 15	1.0- 2.8	5.83 1.19 17	3.5- 7.9	22.1 10.3 19	9- 51
4	5.70 0.46 19	5.0- 6.7	140.1 3.41 19	136- 148	97.8 6.62 19	87- 108	10.15 1.04 19	8.4- 11.8	1.41 0.62 18	0.6- 2.9	5.49 1.24 18	3.4- 8.1	21.7 10.6 19	9- 52
8	5.46 0.50 19	4.8- 6.2	140.4 4.74 19	136- 148	98.1 6.79 19	88- 110	9.78 0.83 19	7.8- 10.8	1.52 0.49 18	0.9- 2.4	5.39 1.27 18	3.0 7.9	24.2 8.6 19	9- 59
12	5.34 0.50 19	4.7- 6.3	141.8 3.91 19	134- 152	96.4 7.17 19	85- 110	9.55 0.87 19	7.8- 10.8	1.35 0.49 18	0.5- 2.3	5.41 1.20 18	3.2- 7.7	26.5 12.49 19	6- 60
18	5.39 0.60 19	4.5- 6.4	140.8 5.62 19	130- 148	97.8 6.67 19	86- 108	9.45 1.11 19	7.6- 11.7	1.27 0.52 17	0.6- 2.7	5.90 1.32 19	3.4- 8.8	30.0 10.6 19	15- 57
24	5.20 0.79 19	3.9- 6.7	140.7 5.00 19	132- 148	97.4 5.91 19	89- 108	9.45 0.93 19	8.1- 11.2	1.24 0.59 18	0.6- 2.7	6.34 1.01 19	4.2- 8.8	32.0 11.45 19	17- 57

TABLE 10. (CONTINUED)

Hours after birth	K	Na	Cl	Ca	Mg	Phosphorus	Urea
48	5.10 0.69 17	141.6 5.06 148 17	98.7 5.50 110 18	9.19 0.70 10.5 16	1.30 0.48 0.6- 15	7.09 1.44 4.5- 17	37.7 20.84 14- 18
72	5.00 0.96 18	142.6 4.25 152 18	98.4 5.54 108 18	9.72 1.09 8.2- 17	1.30 0.54 0.6- 16	7.11 1.50 4.8- 18	39.7 22.24 8- 18
96	4.70 0.85 18	142.4 4.54 148 18	99.7 5.31 109 18	9.74 0.79 8.0- 17	1.32 0.46 0.7- 16	6.44 0.97 4.9- 18	36.8 26.16 9- 18

TABLE 11. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea					
Acharya (1962)												
0 hr	6.55 1.59 14	138.0 9.1 14	120- 150	100.92 7.18 14	9.53 1.8 10	8.6- 12.2	1.54 0.56 10	0.5- 2.6	6.35 1.84 14	4.0- 10.8	31.21 10.24 14	16- 53
1 hr	6.72 1.12 15	139.47 10.8 16	121- 148	101.81 5.87 16	9.29 0.83 12	8.3- 10.8	1.48 0.67 12	0.1- 2.3	7.15 1.96 16	4.0- 11.0	27.37 7.8 16	15- 45
3 hr	7.04 1.26 12	138.46 12.3 15	117- 148	102.5 4.18 14	8.7 0.9 11	7.8- 10.4	1.5 0.31 11	1.2- 1.9	7.23 1.36 16	3.8- 11.1	27.73 9.2 15	17- 47
5 hr	6.84 1.45 13	138.25 8.36 16	120- 160	101.81 6.81 16	8.57 0.49 12	7.6- 9.8	1.44 0.36 12	0.8- 2.6	6.98 1.91 16	4.0- 11.0	31.68 14.7 16	16- 83
7 hr	6.9 1.52 14	138.37 7.65 16	118- 151	101.4 8.2 15	8.37 0.9 12	7.2- 10.3	1.40 0.50 11	0.4- 2.1	6.67 1.57 16	4.3- 9.6	28.56 6.4 16	21- 42
11 or 12 hr	6.73 0.86 14	139.43 10.77 16	121- 148	102.37 6.55 16	8.37 0.87 12	7.1- 10.2	1.44 0.30 12	0.8- 1.7	6.41 1.95 16	4.2- 10.3	33.25 7.83 16	22- 46
18 hr	6.67 0.97 16	139.43 7.68 16	125- 150	101.81 6.82 16	7.97 0.81 12	6.8- 9.5	1.55 0.51 12	1.0- 2.8	6.65 1.7 16	4.0- 10.5	37.62 7.5 16	25- 56
24 hr	6.51 1.61 16	139.93 4.66 16	125- 154	102.68 7.78 16	7.83 0.82 12	6.2- 9.5	1.49 0.48 12	1.1- 2.0	6.78 1.48 16	4.2- 9.5	41.75 10.32 16	26- 55

TABLE 11. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
36 hr	6.46	140.86	103.8	7.99	1.43	6.81	50.93
	0.89 15	157	9.9 15	1.1 11	0.46 10	1.71 15	16.1 15
48 hr	6.21	141	103.12	7.94	1.65	6.62	49.1
	0.82 16	127	8.36 16	0.73 12	0.52 12	1.93 16	15.71 16

FIGURE 4.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM,
INORGANIC PHOSPHORUS, TOTAL PROTEIN AND BLOOD UREA
CONCENTRATIONS (MEAN) OF FULL-TERM NORMAL INFANTS
(• ——— •) AND INFANTS DELIVERED BY DIFFICULT
OBSTETRIC PROCEDURES (▼ — — — ▼).

The scale is reduced after 18 hours.

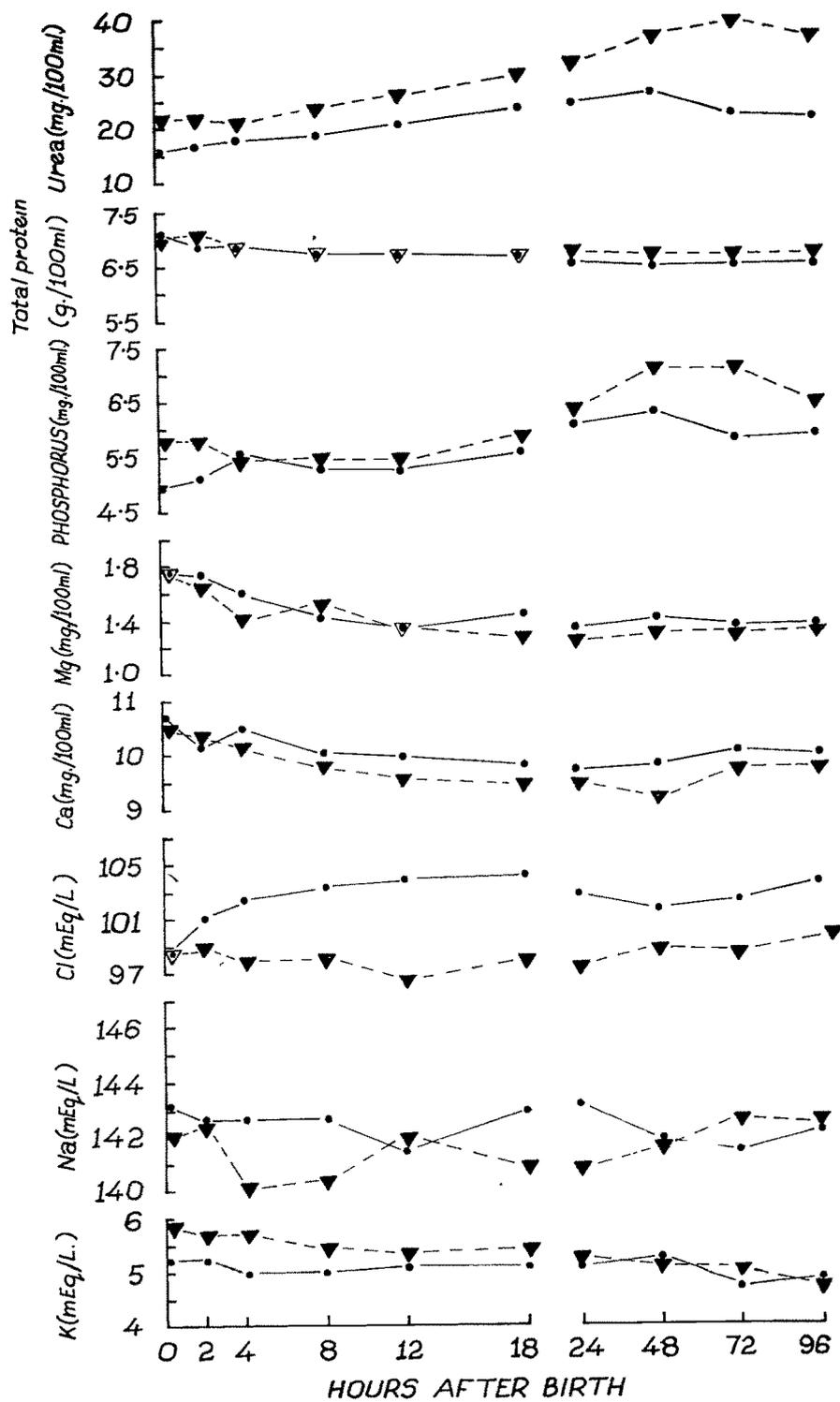


Figure 4.

HEALTHY PREMATURE INFANTS

ACID-BASE STATUSRESULTS:

This group includes 10 infants. The results of acid-base determinations are expressed as mean, standard deviation, range and number in Table 12. The mean changes are expressed graphically in Fig.5.

pH: The mean pH value at birth is 7.24 and remains lower than that of the normal group throughout the period of observation. The difference is significant at 24 hours of age ($t = 2.27, P < 0.05$).

pCO₂: The mean pCO₂ value is higher than that of the normal group for the period of observation except at 24 hours.

BE: The mean base excess concentration is -7.7 mEq/L. at birth which rises gradually to -2.9 mEq/L. at 48 hours and does not attain normal value. The mean concentrations during the period of observation are lower than those of the normal group and a significant difference is found at 24 hours ($t = 2.23, P < 0.05$).

Standard HCO₃: The mean concentration of this parameter is lower as compared to that of the normal group throughout the period of 96 hours after birth.

Actual HCO₃: Much variation is not observed in the mean concentration of the present and the normal group.

H₂CO₃: The mean carbonic acid concentration follows a trend similar to that of pCO₂.

HCO₃/H₂CO₃: The ratio in this group is lower than that of the normal group throughout the period of observation. Significant difference is found at 24 and 48 hours after birth (t = 3.0, P < 0.01, t = 3.0, P < 0.01 respectively).

The course of changes in the acid-base pattern of the same infant of this group is demonstrated in Table 12-A.

The infants show respiratory and non-respiratory acidosis at birth. The respiratory acidosis is corrected after the age of eight hours. By this period, 80 per cent of the infants are in a state of non-respiratory acidosis, 10 per cent in respiratory acidosis and the remaining are in the normal acid-base equilibrium. A gradual correction in the non-respiratory acidosis and improvement in the normal acid-base equilibrium is seen during eight to 18 hours after birth. This is accompanied by an onset of respiratory basosis. Deterioration in the acid-base pattern is found at 24 hours of age with an increased incidence of the non-respiratory acidosis found in 60 per cent of the infants. This is evidenced by significant lower values of pH, BE and HCO₃/H₂CO₃ of this group as compared to those of the normal infants at 24 hours. It is seen from the Table 12-A that 40 to 50 per cent of the infants are in non-respiratory acidosis between 48 to 96 hours of age. The 10 to 20 per cent incidence of respiratory basosis seen between 18 to 48 hours of age in this group is the lowest amongst all the groups studied.

Representative data from the literature on acid-base

status during the early neonatal period is summarized in Table 13.

Biochemical status of the healthy premature infants in the first 48 hours of life was studied by Yu et al. (1965). These workers grouped premature infants into two categories, one weighing less than 1500 G. and the other between 1500 and 2500 G. at birth. The small premature infants attained the normal acid-base status more rapidly than the bigger infants. The heavier infants needed 48 hours to reach a pH of 7.40, while the smaller reached the same value by 12 hours after birth. However, pH value of more than 7.37 was not attained in the present group. The $p\text{CO}_2$ of the heavier weight group remained higher than that of the other group. The trend in both the groups showed a slow fall from the cord blood level. The smaller premature infants had higher values of standard bicarbonate than those of larger infants upto the sixth hour of life.

Bucci et al. (1965) conducted a study of acid-base status of normal premature infants in the first week of life. Combined acidosis was found at the age of three to six hours in the group of infants weighing more than 1250 G. at birth. Significant changes were found in pH ($P < 0.001$) and BE ($P < 0.025$) from the age of three to six and seven to 12 hours. The average values between 13 to 48 hours of age indicated a mild compensated metabolic acidosis. A significant ($P < 0.025$) decrease of the BE occurred, almost compensated by a decrease of the $p\text{CO}_2$, from the second to third day of life. From the third to the fourth or fifth day, significant increases in the

BE ($P < 0.025$) and $p\text{CO}_2$ ($P < 0.005$) were observed.

Malan et al. (1965) carried out a serial acid-base study in the normal premature infants during the first 72 hours of life. A correlation in the observations of these workers and those in the present series can be seen in: (i) the persistence of respiratory acidosis and a higher level of $p\text{CO}_2$ in the healthy premature infants, (ii) contribution of the non-respiratory acidosis as the largest single group, (iii) maximum number of infants in the non-respiratory acidosis at 24 hours of age, (iv) persistence of the non-respiratory acidosis in about 40 to 50 per cent of infants even after 48 hours of age.

Malan et al. (1966) also reported acid-base determinations in the normal premature infants during the first two months of life. The mean values for pH, base excess, buffer base and standard bicarbonate showed a gradual movement towards the non-respiratory acidosis from fourth to 14th day. During the third week, the values remained stationary and thereafter showed a rise. Majority of the babies had non-respiratory acidosis, usually uncompensated even between 22nd to 60th days of life.

A study of birth weight and acid-base equilibrium in the first days of life in the premature infants was carried out by Severi et al. (1970). These authors found the persistence of non-respiratory acidosis throughout the period of seven days. An initial correction in this stage upto three days and deterioration thereafter were observed by them.

DISCUSSION:

It would be observed from the results of the present series that there is an increase in the incidence and duration of the respiratory acidosis in this group than that of the normal group. Similar findings have been reported by Kildeberg (1964), Bucci et al. (1965) and Malan et al. (1965). In the absence of a demonstrable pulmonary disease, the higher incidence and magnitude of respiratory acidosis in the premature infants can only be attributed to a poor sensitivity of the respiratory centre in response to hypoxia. The maximum number of infants in the premature group (60 per cent) show non-respiratory acidosis at 24 hours, in contrast to only 12 per cent of the infants in the normal group. The ability of the neonatal kidney is limited to establish the adult pH gradient between blood and urine and to excrete H⁺ ions into the urine as there is a deficiency of urinary buffer substances (Mc Cance and Hatemi, 1961). These limitations of the kidney function would be more marked in the premature infants. Furthermore, an increased tissue catabolism is observed in these infants which is evidenced by a rise in the plasma magnesium and blood urea concentrations at 24 hours (Fig.6). Thus the diminished renal function coupled with an increased tissue catabolism would account for higher incidence of the non-respiratory acidosis seen in the premature infants. The subsequent persistence of the non-respiratory acidosis beyond 24 hours could also be due to these factors.

TABLE 12. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.37 0.033 10	37.8 2.64 10	-2.9 1.88 10	21.7 1.63 10	21.2 1.26 10	1.13 0.10 10	18.7 1.22 10
72	7.32- 7.41	33.0- 40.0	-6.5 -1.0	19.3- 23.2	18.0- 23.0	0.99- 1.20	16.5- 20.2
96	7.37 0.033 10	38.3 3.16 10	-3.0 2.43 10	21.7 2.17 10	21.3 2.42 10	1.15 0.06 10	18.43 1.71 10
	7.30- 7.42	34.0- 41.0	-7.5 +1.0	18.4- 25.5	18.0- 26.0	0.98- 1.23	15.3- 20.0
	7.36 0.047 10	38.1 2.54 10	-3.4 2.55 10	21.4 3.79 10	21.0 2.03 10	1.14 0.10 10	18.41 1.82 10

TABLE 13.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE AND ACTUAL BICARBONATE CONCENTRATIONS OF HEALTHY PREMATURE INFANTS. (REPRESENTATIVE DATA FROM LITERATURE)

Author (Year) Age	Sample	pH		pCO ₂ mm. Hg.		BE mEq/L.		Std. HCO ₃ mEq/L.		Actual HCO ₃ mEq/L.	
		Mean SD+ No.	Range	Mean SD+ No.	Range	Mean SD+ No.	Range	Mean SD+ No.	Range	Mean SD+ No.	Range
Pincus et al. (1956)											
0-24 hr	0	7.34 0.09 25	-	35.0 10.0 25	-	-	-	-	-	20.0 4.0 25	-
Reardon et al. (1960)											
48 hr	A	7.31	-	38.9	-	-6.3	-	-	-	19.4	-
Bucci, Scalamandre, Savigoni & Mendicini (1965)											
3-6 hr	0	7.32 0.07 11	-	43.7 21.5 9	-	-4.82 2.7 12	-	-	-	-	-
7-12 hr	0	7.359 0.05 21	-	38.7 9.9 16	-	-3.87 2.7 22	-	-	-	-	-
13-24 hr	0	7.391 0.05 46	-	38.3 7.5 41	-	-2.44 2.8 48	-	-	-	-	-
2 day	0	7.385 0.05 49	-	38.9 7.4 43	-	-2.32 2.0 54	-	-	-	-	-

TABLE 13. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std.HCO ₃	Actual HCO ₃
3 day	C	7.376	37.7	-3.39	-	-
		0.04 39	8.7 39	2.21 41	-	-
4-5 day	C	7.364	42.3	-1.86	-	-
		0.04 41	5.7 40	2.2 40	-	-
Malan et al. (1965)	C	7.33	42.8	-3.7	21.2	21.8
		0.06 17	6.8 17	2.9 17	1.8 17	2.9 17
	C	7.35	43.4	-2.3	22.2	22.9
		0.04 17	8.2 17	2.6 17	2.2 17	3.7 17
	C	7.36	40.9	-2.1	22.5	23.3
		0.04 17	9.6 17	3.0 17	1.4 17	4.3 17
	C	7.33	38.4	-2.3	22.1	21.7
		0.03 17	6.4 17	1.8 17	1.6 17	2.6 17
	C	7.39	35.8	-2.6	21.9	20.8
		0.04 17	4.0 17	1.8 17	1.6 17	1.9 17
	C	7.37	39.5	-2.7	21.9	21.6
		0.05 17	6.1 17	1.2 17	1.7 17	1.6 17
C	7.37	36.8	-3.3	21.4	20.6	
	0.05 17	5.2 17	2.9 17	2.0 17	2.7 17	

TABLE 13. (CONTINUED)

Author (Year) Age	Sample	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃
Malan et al. (1966)	0	7.33	39.9	-4.8	20.4	19.9
		0.04	4.0	2.2	1.2	0.4
5-7 day	0	7.27-	31.2-	-6.9	18.2-	17.9-
		7.45	43.0	-3.9	23.5	21.2
	0	7.32	39.4	-6.8	19.5	19.5
		0.03	10.2	2.4	3.0	4.8
Severi et al. (1970)	0	7.23-	26.1-	-10.1	16.2-	14.8-
		7.40	63.0	-1.9	28.5	33.4
2-3 hr	0	7.297	43.73	-6.30	19.45	-
		0.047	8.05	2.36	1.59	-
24 hr	0	18	18	18	18	-
		7.351	34.31	-5.73	19.96	-
3 day	0	0.038	8.05	2.42	1.54	-
		17	17	17	17	-
5 day	0	7.367	36.79	-3.70	21.25	-
		0.050	6.24	2.51	1.79	-
7 day	0	18	18	18	18	-
		7.345	37.01	-4.80	20.22	-
	0	0.057	4.69	3.47	2.21	-
		18	18	18	18	-
	0	7.340	38.57	-5.32	20.35	-
		0.062	6.96	3.98	2.62	-
		17	17	17	17	-

A = Arterial

C = Capillary

FIGURE 5.

BLOOD pH, pCO_2 , BASE EXCESS, STANDARD BICARBONATE,
ACTUAL BICARBONATE, CARBONIC ACID AND
BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF
FULL-TERM NORMAL INFANTS (•————•) AND HEALTHY
PREMATURE INFANTS (○———○).

The scale is reduced after 18 hours.

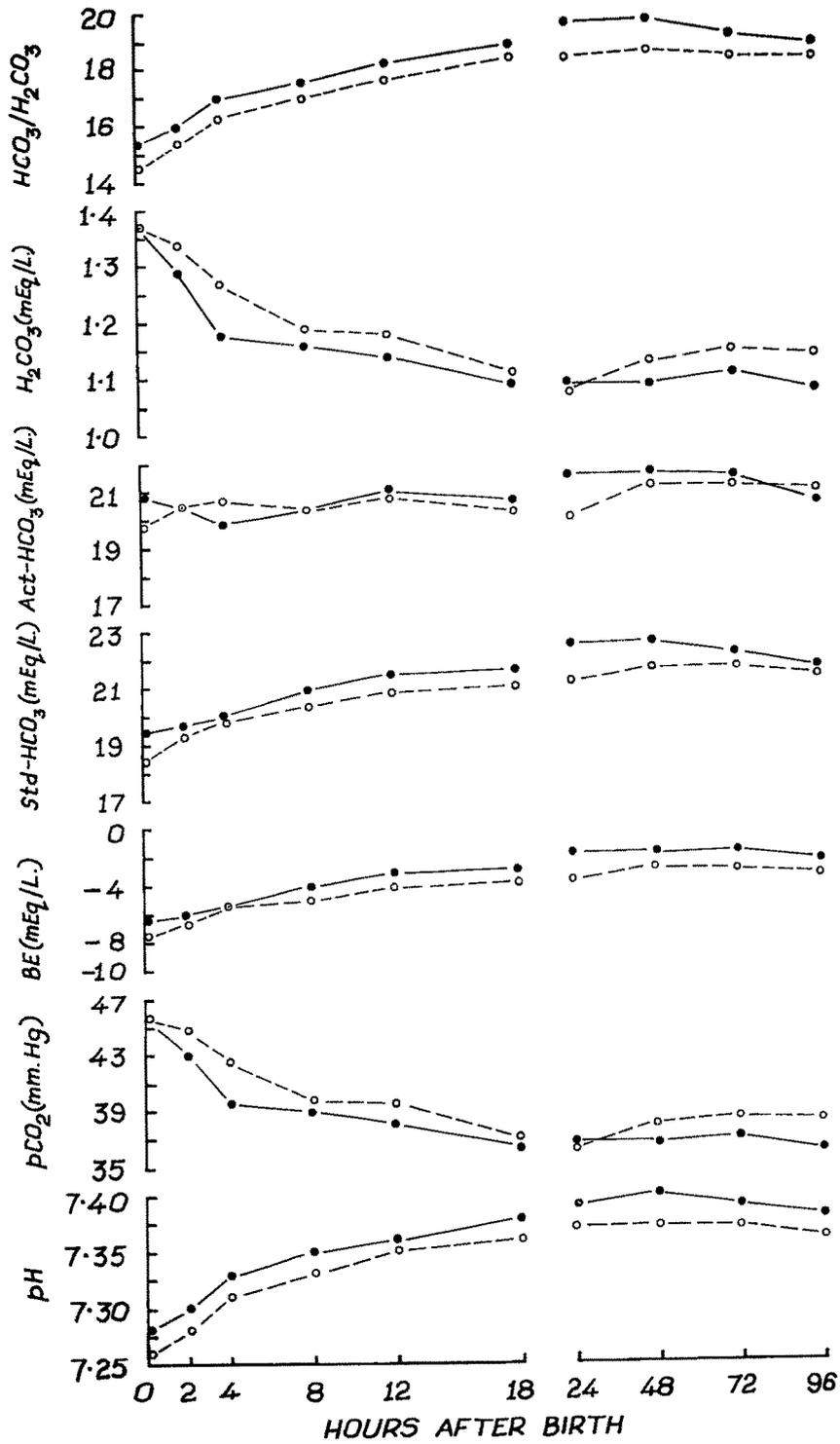


Figure 5.

PLASMA ELECTROLYTE STATUS

RESULTS:

The number of infants studied in this group is 10. The results of their electrolyte determinations are expressed as mean, standard deviation, range and number in Table 14, while Fig.6 shows the behaviour of their mean concentrations.

Plasma Potassium: The behaviour of mean concentration of plasma potassium shows a gradual fall after birth except at 12 hours. The values are higher than those of the normal group upto four hours and remain lower from 18 hours onwards.

Plasma Sodium: The mean concentration of plasma sodium is found higher than that of the normal group during the whole period.

Plasma Chloride: The mean concentration of the plasma chloride is seen to be lower than that of the normal group except at and 48 hours after birth. A statistically significant ($t = 2.15$, $P < 0.05$) decrease is found at 18 hours of age.

Plasma Calcium: The mean concentration of the plasma calcium is lower than that of the normal group during the period of 96 hours, but shows a similar trend. However, the decrease is significant at 48, 72 and 96 hours of age ($P < 0.05$, < 0.05 and < 0.001 respectively).

Plasma Magnesium: An increased concentration of plasma magnesium as compared to that of the normal group is found

during the 96 hour period after birth. The values are significantly higher at 12 and 72 hours of age ($t = 2.2$, $P < 0.05$ and $t = 3.23$, $P < 0.01$ respectively).

Plasma Inorganic Phosphorus: The mean concentration of plasma inorganic phosphorus is higher than that of the normal group till four hours after birth. Then the values are lower than those of the normal group upto 48 hours of age. The highest inorganic phosphorus concentration coincides with the lowest concentration of plasma calcium at 96 hours.

Plasma Total Protein: The highest value, 6.0 g./100 ml. at birth falls upto the lowest value, 5.53 g./100 ml. at 24 hours. The fall is about 8 per cent. The mean total protein concentration is significantly lower than that of the normal group throughout the period of observation ($P < 0.001$ from birth to 72 hours and $P < 0.01$ at 96 hours). The mean total protein of the infants at birth is lower than that of the mothers at delivery.

Blood Urea: The mean blood urea concentration is lower upto 12 hours as compared to that of the normal group. The level from 24 hours onwards is higher than that of the normal group without any significance.

Representative data from the literature on the electrolyte status during the early neonatal period are summarized in Table 15 and 17.

A study of biochemical status of the healthy premature infants in the first 48 hours of life was carried out by Yu et al. (1965). Serum potassium levels in the cord blood were high, ranging from 5.0 to 10.2 mEq/L. Serum potassium

concentration showed a rise after three hours in 50 per cent of the babies. Infants of low weight group had cord blood sodium higher (142-163 mEq/L.) than those of the heavier ones (132 mEq/L.). The serum sodium slowly rose by 5 mEq/L. in all the babies at 48 hour of age. The serum chloride values were within the normal adult range throughout the period of study. The plasma calcium levels in the cord blood of premature infants were high (10.6 mg./100 ml.) and fell to a minimum level of 8.6 mg./100 ml. at 18 to 24 hours. The trend in serum magnesium level was very similar to that of serum calcium, with a high cord value, a subsequent fall and then recovery. The concentration was 2.1 mg./100 ml. at 48 hours after birth. Blood inorganic phosphate levels, did not correlate with the weights of the babies. There was an upward trend during the period of study till 48 hours. Plasma protein concentration showed a fall upto six hours. This was followed by a rise. Blood urea values were within the normal adult range and showed an increase within six to 24 hours after birth.

Tsang and Oh (1970) found magnesium concentration of cord blood to be lower than that of the first week of life.

Jukarainen (1971) studied the behaviour of plasma calcium, magnesium and phosphorus concentrations for the first five days of life in 30 pre-term neonates with gestation ages from 28 to 36 weeks. The mean magnesium varied within very narrow

limits. The highest value of 2.3 mg./100 ml. at 82 hours was 8.5 per cent higher than the lowest value of 2.1 mg./100 ml. at 28 hours, while calcium concentration showed an inverse pattern. The mean highest value of 9.0 mg./100 ml. at the age of 0 to 2 hour fell distinctly to 6.8 mg./100 ml. at the age of 44 hours. It was 24.5 per cent fall. The maximum value of plasma inorganic phosphorus of 7.9 mg./100 ml. at 44 hours after birth coincided with the minimum calcium levels. This maximum value was 21.5 per cent higher than the value at the age of 6 hours.

DISCUSSION:

The initial high values of plasma potassium could be on account of respiratory acidosis, the disappearance of which is accompanied by a fall in plasma potassium. The plasma potassium does not show any significant change thereafter to comment. The initial plasma sodium level of 146.5 mEq/L. is higher than that of the normal group. The gradual fall observed till eight hours may be due to a deficiency of mineralocorticoids and/or acidosis. Subsequently a gradual increase is seen till 48 hours which could be explained on the basis of dehydration. Yu et al. (1965) are of the same opinion for a slow rise in the plasma sodium concentration during similar period. Thereafter, plasma sodium tries to attain a normal equilibrium. The plasma chloride concentrations do not show much change to comment upon. The plasma calcium concentration shows a falling trend. This could be due to similar mechanisms of hypoparathyroidism, release of thyrocalcitonin and mineralization of bone as accounted for in the normal infants. Furthermore, the persistence of acidosis in the premature infants would contribute to a higher magnitude of fall in the plasma calcium as seen in the present group. An initially high level of plasma inorganic phosphorus is on account of a greater degree of acidosis observed in these infants. A slow and sustained rise (20 per cent) seen after eight hours till 96 hours is a change reciprocal to that of plasma calcium.

The late rise in the plasma magnesium and blood urea concentrations (32 and 195 per cent respectively) signify an increased rate and magnitude of tissue breakdown occurring in the premature infants. It appears that the protein synthesis is deficient in the premature liver as evidenced by the lower plasma protein level in this group of infants.

TABLE 14.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF HEALTHY PREMATURE INFANTS. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
0	5.58 0.57 10	4.6- 6.5	146.5 6.03 8	134- 154	101.6 6.35 8	93- 109	10.31 1.88 8	8.9- 12.3	1.98 0.98 6	1.1- 3.6	5.73 1.49 8	3.8- 7.7	12.1 6.85 8	3- 24
2	5.44 1.21 10	3.8- 8.1	144.2 5.37 10	134- 150	100.8 5.15 10	91- 107	10.11 0.62 10	9.1- 10.6	1.84 0.88 9	0.4- 3.4	5.63 2.04 10	2.3- 8.6	13.0 5.81 10	4- 24
4	5.41 1.01 10	4.0- 7.2	143.2 5.43 10	136- 152	101.3 4.87 10	94- 109	9.98 0.51 10	9.3- 11.0	1.72 0.73 9	0.6- 3.0	5.22 1.39 10	2.9- 6.8	13.4 6.38 10	7- 25
8	4.97 1.24 10	3.5- 7.2	142.6 2.99 10	136- 146	101.4 5.73 10	91- 109	9.83 0.59 10	8.6- 10.4	1.53 0.56 9	0.8- 2.4	5.07 1.50 10	2.3- 7.3	16.5 6.62 10	7- 27
12	5.45 0.54 10	4.4- 6.3	143.6 4.30 10	134- 148	100.2 4.56 10	93- 107	9.76 0.76 10	8.2- 11.1	1.78 0.78 9	0.7- 3.0	5.12 1.43 10	2.7- 6.8	20.7 9.28 10	6- 35
18	4.85 0.64 10	3.6- 5.7	143.4 5.49 10	134- 152	100.0 3.65 10	93- 107	9.33 0.93 10	8.1- 10.6	1.71 0.92 9	0.7- 3.7	5.41 1.07 10	3.9- 7.1	24.8 12.04 10	6- 46
24	4.81 0.62 10	3.9- 5.7	144.2 4.92 10	134- 152	101.0 4.05 10	93- 107	9.37 0.69 10	8.3- 10.5	1.73 0.78 9	0.9- 3.1	5.63 1.25 10	3.1- 7.0	31.9 18.65 10	6- 67

TABLE 14. (CONTINUED)

Hours after birth	K	Na:	Cl	Ca:	Mg:	Phosphorus	Urea
48	4.72 0.90 9	144.8 4.33 10	102.0 2.94 10	9.18 0.53 10	1.81 0.84 9	5.82 1.16 10	35.7 20.5 10
72	4.63 0.92 8	142.8 6.48 10	100.0 3.80 10	9.33 0.91 10	2.02 0.56 8	5.84 1.34 10	34.3 21.65 10
96	4.67 1.10 10	142.2 5.29 10	103.1 4.38 10	9.13 0.97 10	1.88 0.80 9	6.01 1.55 10	29.9 20.15 10

TABLE 15. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
					Mg		
73 hr	-	-	-	-	2.32 0.35 79	-	-
97 hr	-	-	-	-	2.37 0.35 75	-	-
Jukareinen (1971)					mEq/L.		
0-2 hr	-	-	-	4.5 0.6 14	1.91 0.31 14	7.4 2.2 15	-
2-8 hr	-	-	-	4.2 0.7 14	1.80 0.34 12	6.5 2.9 12	-
8-16 hr	-	-	-	3.9 0.7 25	1.88 0.34 22	6.9 2.2 22	-
16-24 hr	-	-	-	3.7 0.7 26	1.88 0.39 24	7.4 2.9 22	-
24-32 hr	-	-	-	3.5 0.7 25	1.78 0.27 23	7.1 1.7 25	-
32-40 hr	-	-	-	3.5 0.7 24	1.91 0.29 21	7.9 1.8 22	-
40-48 hr	-	-	-	3.4 0.6 27	1.84 0.29 21	7.9 1.6 22	-

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TABLE 15. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
48-56 hr	-	-	-	3.6 0.8 24	1.85 0.28 23	7.0 1.7 23	-
56-64 hr	-	-	-	3.5 0.7 25	1.86 0.31 21	6.9 2.0 21	-
64-76 hr	-	-	-	3.6 0.6 27	1.80 0.28 22	6.7 2.0 23	-
76-88 hr	-	-	-	3.8 0.6 25	1.93 0.39 19	6.7 2.2 20	-
88-100 hr	-	-	-	3.9 0.5 21	1.90 0.36 17	6.3 1.6 18	-
100-128 hr	-	-	-	4.0 0.5 23	1.85 0.33 21	6.1 2.0 20	-

FIGURE 6.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM,
INORGANIC PHOSPHORUS, TOTAL PROTEIN AND BLOOD UREA
CONCENTRATIONS (MEAN) OF FULL-TERM NORMAL INFANTS
(• ——— •) AND HEALTHY PREMATURE INFANTS (○ — — — ○).

The scale is reduced after 18 hours.

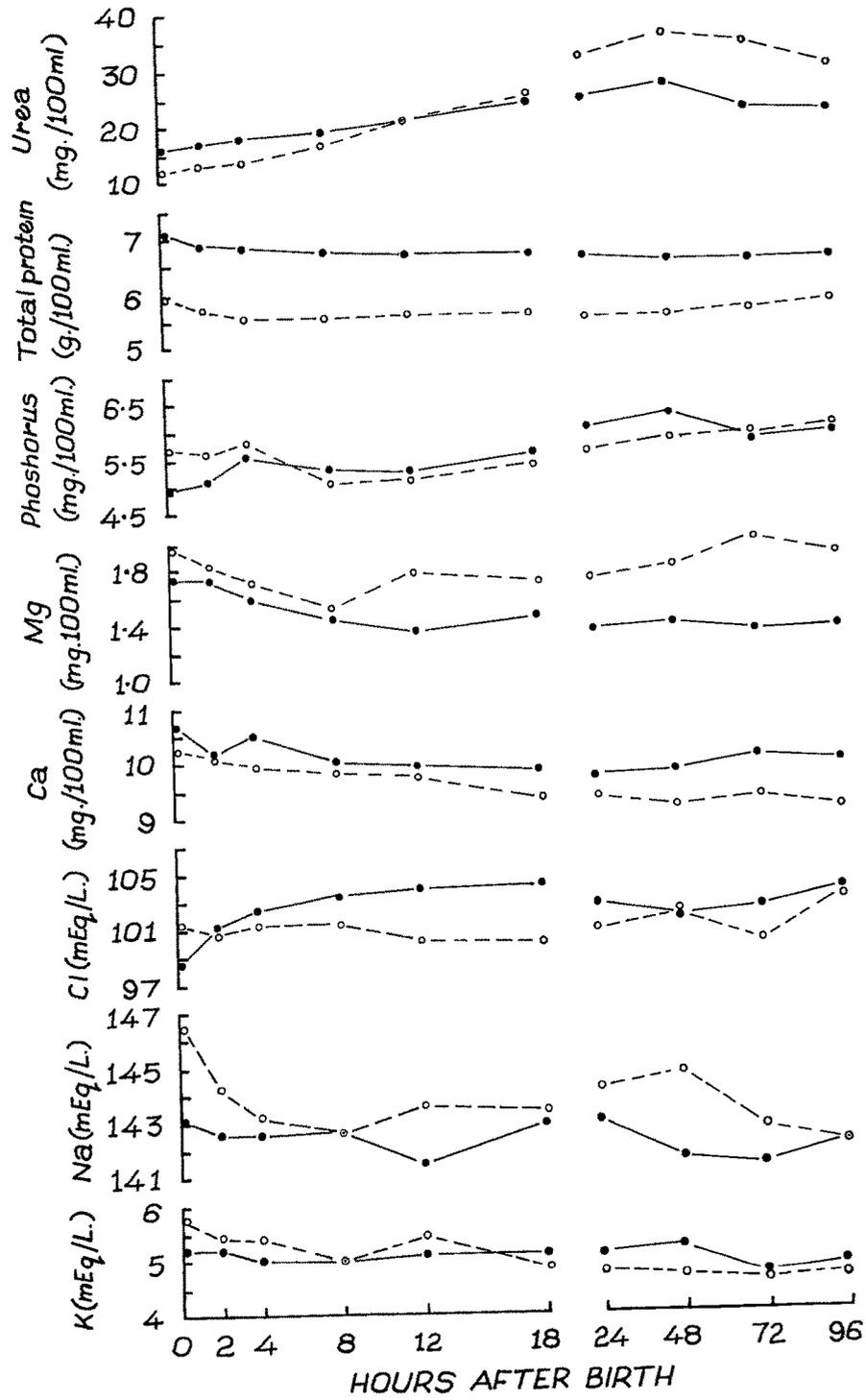


Figure 6.

PLASMA PROTEIN FRACTIONS

RESULTS:

In the present group, electrophoretic separation of plasma protein is done for a period of 96 hour after birth in nine infants and their mothers at delivery. The results are expressed as mean, standard deviation, range and number of determinations in Table 16.

Plasma Albumin: The mean concentration of plasma albumin is below the normal adult range and significantly lower than that of the normal group at zero, two, four, eight, 12, 18, 24, 48, 72 and 96 hours after birth ($P < 0.02$, < 0.01 , < 0.02 , < 0.01 , < 0.01 , < 0.01 , < 0.02 , < 0.01 , < 0.02 and < 0.05 respectively). Plasma albumin concentration does not show much variation during the whole period. The maternal plasma albumin concentration is much lower than that of the infant at birth.

Plasma Alpha₁, Alpha₂ and Beta: The mean concentration of these globulin fractions in the present series are lower than those of the normal group. However, a significant decrease ($t = 2.05$, $P < 0.05$) in the beta fraction is found at four hours of age.

Plasma Gamma Globulin: The concentration of plasma gamma globulin is 1.20 g./100 ml. at birth and remains lower than this for the rest of the period. The values are significantly lower than the normal group at birth, two, four, eight and 24 hours ($P < 0.05$, < 0.02 , < 0.01 , < 0.01 and < 0.05 respectively).

Concentrations of the neonate's globulin fractions are much lower than those of the mother.

Representative data from the literature on serum/plasma protein electrophoretic patterns during the early neonatal period are summarized in Table 17.

Saito et al. (1956) studied plasma protein patterns in the premature infants of varying weights on the first day of life and reported a positive correlation between the plasma albumin and globulin concentrations and the birth weight of the babies.

DISCUSSION:

The lower plasma albumin concentration found in this group could be accounted by the functional immaturity of liver which results in a diminished synthesis of albumin. This is in agreement with the views of Khalil et al. (1968). The changes observed in the plasma α_1 , α_2 and beta fractions show a similar trend as found in the normal group and are due to the same mechanism. The gamma globulin value in the premature infant is significantly lower ($P < 0.05$) than that of the normal group at birth. It appears that the peak concentration via placental transfer is not attained in these infants on account of the pre-term birth.

TABLE 16.
 PLASMA TOTAL PROTEIN AND PROTEIN FRACTIONS (g./100 ml.) OF HEALTHY PREMATURE INFANTS AND
 THEIR MOTHERS AT DELIVERY. (PRESENT SERIES)

Hours after birth	T. Protein Mean Range SD+ No.	Albumin			Globulin							
		Mean SD+ No.	Range	Mean SD+ No.	Alpha ₁		Alpha ₂		Beta		Gamma	
					Mean SD+ No.	Range	Mean SD+ No.	Range	Mean SD+ No.	Range	Mean SD+ No.	Range
Mothers at del- ivery	6.36 0.63 8	2.37 (37.3) 0.12 8	1.90- 3.07	0.35 (5.5) 0.09 8	0.20- 0.46	0.74 (11.6) 0.18 8	0.53- 1.10	1.13 (17.8) 0.18 8	1.02- 1.40	1.77 (27.8) 0.33 8	1.28- 2.13	
0	6.0 0.67 8	3.70 (61.6) 0.55 7	2.95- 4.70	0.21 (3.5) 0.09 7	0.10- 0.35	0.35 (5.7) 0.13 7	0.23- 0.62	0.54 (8.8) 0.16 7	0.39- 0.88	1.20 (20.4) 0.37 7	0.53- 1.53	
2	5.70 0.70 10	3.56 (62.5) 0.54 9	2.80- 4.45	0.19 (3.3) 0.09 9	0.08- 0.38	0.33 (5.7) 0.19 9	0.17- 0.66	0.44 (7.6) 0.15 9	0.29- 0.79	1.18 (20.9) 0.36 9	0.57- 1.58	
4	5.60 0.71 10	3.55 (63.3) 0.58 8	2.85- 4.45	0.16 (2.8) 0.07 8	0.11- 0.32	0.30 (5.3) 0.12 8	0.19- 0.55	0.46 (7.9) 0.20 8	0.30 0.93	1.13 (20.7) 0.26 8	0.81- 1.47	
8	5.60 0.58 10	3.54 (63.5) 0.43 9	3.00- 4.15	0.17 (3.1) 0.09 9	0.07- 0.32	0.32 (5.7) 0.14 9	0.21- 0.61	0.49 (8.6) 0.16 9	0.28- 0.80	1.08 (19.1) 0.28 9	0.69- 1.42	
12	5.60 0.57 10	3.55 (63.1) 0.58 9	2.60- 4.45	0.16 (3.0) 0.08 9	0.07- 0.26	0.31 (5.5) 0.09 9	0.17- 0.44	0.47 (8.3) 0.18 9	0.26- 0.76	1.12 (20.1) 0.38 9	0.63- 1.60	

TABLE 16 (CONTINUED)

Hours after birth	T. Protein	Albumin	Globulin			Gamma
			Alpha ₁	Alpha ₂	Beta	
18	5.68	3.67	0.16	0.30	0.43	1.11
	5.0- 0.49 10	2.45- 4.55 0.67 8	0.10- 0.22 0.05 8	0.21- 0.40 0.06 8	0.28- 0.70 0.14 8	0.82- 1.48 0.23 8
24	5.53	3.60	0.17	0.28	0.44	1.04
	4.90- 0.50 10	2.35- 4.50 0.75 9	0.08- 0.23 0.04 9	0.23- 0.33 0.03 9	0.29- 0.78 0.16 9	0.81- 1.54 0.28 9
48	5.60	3.51	0.20	0.30	0.48	1.11
	4.95- 0.46 10	2.70- 4.60 0.66 9	0.12- 0.31 0.06 9	0.19- 0.42 0.08 9	0.27- 0.75 0.15 9	0.70- 1.60 0.33 9
72	5.70	3.48	0.21	0.33	0.50	1.18
	5.05- 0.46 10	2.75- 4.90 0.70 9	0.13- 0.31 0.07 9	0.26- 0.39 0.07 9	0.32- 0.71 0.13 9	0.89- 1.54 0.21 9
96	5.76	3.50	0.20	0.34	0.53	1.20
	5.35- 0.56 10	2.60- 4.75 0.76 9	0.10- 0.28 0.07 9	0.20- 0.45 0.09 9	0.30- 0.80 0.15 9	0.87- 1.66 0.27 9

Figures in the parenthesis indicate percentage of total protein.

TABLE 17. (CONTINUED)

Author (Year) Age	T. Protein	Albumin	Globulin			Gamma
			Alpha ₁	Alpha ₂	Beta	
Khalil et al. (1968):						
Cord	5.67	4.2-	0.28	0.12-	0.68	0.75
	0.81	6.7	0.23	1.12	0.43	0.43
	19	19	19	19	19	19
		2.06-	0.0-	0.12-	0.10-	0.18-
		4.36	0.63	1.12	1.78	1.59

HEALTHY LOW BIRTH WEIGHT INFANTS

ACID-BASE STATUSRESULTS:

The acid-base parameters are studied in 11 infants during the 96 hours of age. The results are expressed as mean, standard deviation, range and number of determinations in Table 18. Their mean concentrations are graphically demonstrated in Fig.7.

pH: The mean pH value at birth is 7.27 which attains lower limits of the normal adult range at 12 hours. The values remain between 7.37 to 7.39 from 24 hours onwards.

pCO₂: The initial mean values of pCO₂ upto two hours are lower than those of the normal group. Higher significant value (t = 2.50, p < 0.05) as compared to that of the normal group is seen only at 18 hours of age.

BE, Standard HCO₃ and Actual HCO₃: The concentrations of these parameters are lower than those of the normal group at birth. Thereafter, a trend similar to that of the normal group is found.

H₂CO₃ and HCO₃/H₂CO₃: The concentrations of these parameters also do not show any significant differences.

The course of changes in the acid-base pattern of the same infant of this group is shown in Table 18-A.

Combined acidosis and non-respiratory acidosis are found at birth. At the age of eight hours, 82 per cent of the infants are in a state of non-respiratory acidosis, 9 per cent

show respiratory acidosis and the rest are in the normal acid-base balance. These changes are in agreement with those of the normal group at eight hours after birth. A quicker and complete recovery from non-respiratory acidosis as compared to that of the normal group is evidenced at 24 hours of age. Further, 45 to 55 per cent of infants remain in the normal acid-base balance between 48 to 96 hours. The incidence of respiratory basosis is seen between 12 to 96 hours of age and its magnitude is higher as compared to the other groups.

DISCUSSION:

The changes observed in the acid-base status of the low birth weight infants during the first eight hours of life are very similar to those seen in the normal full-term infants and could be explained on similar mechanisms. The quicker removal of the non-respiratory acidosis is on account of an effective compensatory mechanism of hyperventilation. The hyperventilation is seen to be continued in a higher percentage of infants leading to the development of respiratory basosis. This continuation of hyperventilation could be due to a much poor sensibilization of the respiratory centre in these infants. Could this be on account of intra-uterine growth retardation?

TABLE 18.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE, CARBONIC ACID AND BICARBONATE/CARBONIC ACID CONCENTRATIONS OF HEALTHY LOW BIRTH WEIGHT INFANTS. (PRESENT SERIES)

Hours after birth	pH		pCO ₂ mm. Hg.		BE mEq/L.		Std. HCO ₃ mEq/L. Plasma		Actual HCO ₃ mEq/L. Plasma		H ₂ CO ₃ mEq/L. Plasma		HCO ₃ /H ₂ CO ₃	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.
0	7.27 0.037 8	7.23- 7.33	43.6 6.7 8	35.0- 50.0	-8.2 1.35 8	-10.0 -6.0	18.3 1.43 8	17.2- 20.0	19.3 2.07 8	17.0- 23.0	1.31 0.19 8	1.05- 1.68	14.9 1.19 8	13.5- 17.0
2	7.30 0.032 11	7.27- 7.35	41.0 4.84 11	33.0- 49.0	-6.5 1.02 11	-8.0 -4.5	19.2 1.21 11	18.5- 20.8	19.6 1.70 11	16.5- 22.2	1.23 0.14 11	0.99- 1.47	16.0 0.77 11	14.8- 18.5
4	7.32 0.032 11	7.29- 7.35	41.2 3.60 11	36.0- 49.0	-5.3 1.46 11	-7.0 -3.0	20.1 0.95 11	18.7- 21.7	20.5 1.61 11	17.8- 22.7	1.24 0.11 11	1.08- 1.47	16.6 1.02 11	15.4- 18.1
8	7.35 0.032 11	7.32- 7.37	39.2 3.57 11	34.0- 46.0	-4.1 1.45 11	-6.0 -1.0	20.9 1.43 11	19.7- 23.2	20.7 1.77 11	18.0- 23.3	1.17 0.14 11	1.02- 1.38	17.6 0.99 11	16.2- 18.7
12	7.36 0.032 11	7.32- 7.39	38.9 4.29 11	32.0- 45.0	-3.4 1.16 11	-5.8 -2.0	21.4 0.74 11	20.0- 22.5	21.1 1.46 11	17.5- 24.0	1.16 0.15 11	0.96- 1.35	18.1 0.52 11	16.2- 19.4
18	7.37 0.032 11	7.33- 7.39	38.7 3.21 11	32.0- 44.0	-2.8 1.41 11	-5.8 -0.5	21.9 0.62 11	19.7- 23.5	21.5 1.60 11	17.5 23.5	1.16 0.10 11	0.96- 1.32	18.5 0.74 11	16.9- 19.6
24	7.38 0.032 11	7.36- 7.40	37.5 4.84 11	32.0- 44.0	-2.1 1.71 11	-4.5 0.0	22.4 0.79 11	20.2- 24.0	21.6 2.45 11	17.7- 25.0	1.13 0.12 11	0.96- 1.32	19.2 0.50 11	18.0- 20.2

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TABLE 18. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.39 0.028 11	36.0 30.0- 3.02 45.0 11	-2.4 -4.5 2.24 +2.0 11	22.2 19.0- 1.20 25.3 11	20.9 16.2- 2.87 22.3 11	1.08 0.91 0.12 1.35 11	19.3 17.5- 0.70 20.2 11
72	7.39 0.021 11	35.9 32.0- 2.48 40.0 11	-2.2 -6.7 1.98 +0.5 11	22.2 19.3- 1.73 24.2 11	21.0 17.2- 2.90 24.5 11	1.08 0.96- 0.07 1.20 11	19.4 17.3- 0.97 20.4 11
96	7.39 0.015 10	36.4 31.0- 5.28 48.0 10	-3.2 -10.4 2.85 -1.0 10	21.4 14.7- 1.84 23.2 10	20.2 16.3- 2.44 23.0 10	1.09 0.90- 0.15 1.44 10	18.8 11.3- 2.70 20.5 10

TABLE 18-A.
COURSE OF ACID-BASE STATUS IN 11 HEALTHY LOW-BIRTH-WEIGHT INFANTS

Hours after birth	Combined acidosis	Respiratory acidosis	Non-respiratory acidosis	Normal	Respiratory alkalosis
0	1 2 5 (37)		3 4 6 8 11 (63)		
2	2 5 (18)		1 3 4 6 7 8 9 10 11 (82)		
4	2 (9)		1 3 4 5 7 8 9 10 11 (82)	6 (9)	
8		9 (9)	1 2 3 4 5 7 8 10 11 (82)	6 (9)	
12		9 (9)	2 4 7 10 (37)	3 6 8 (27)	1 5 11 (27)
18			2 4 7 (27)	1 3 6 9 10 11 (55)	5 8 (18)
24				1 2 6 7 9 10 11 (64)	3 4 5 8 (36)
48			2 5 (18)	6 7 9 10 11 (46)	1 3 4 5 8 (36)
72			2 (9)	3 4 6 7 9 11 (55)	1 5 8 10 (36)
96	2 (10)			1 3 7 9 11 (50)	4 5 8 10 (40)

Figures shown in the circle indicate per cent of infants.

FIGURE 7.

BLOOD pH, pCO_2 , BASE EXCESS, STANDARD BICARBONATE,
ACTUAL BICARBONATE, CARBONIC ACID AND
BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF
FULL-TERM NORMAL INFANTS (• ————— •) AND HEALTHY
LOW BIRTH WEIGHT INFANTS (▲ - - - - - ▲).

The scale is reduced after 18 hours.

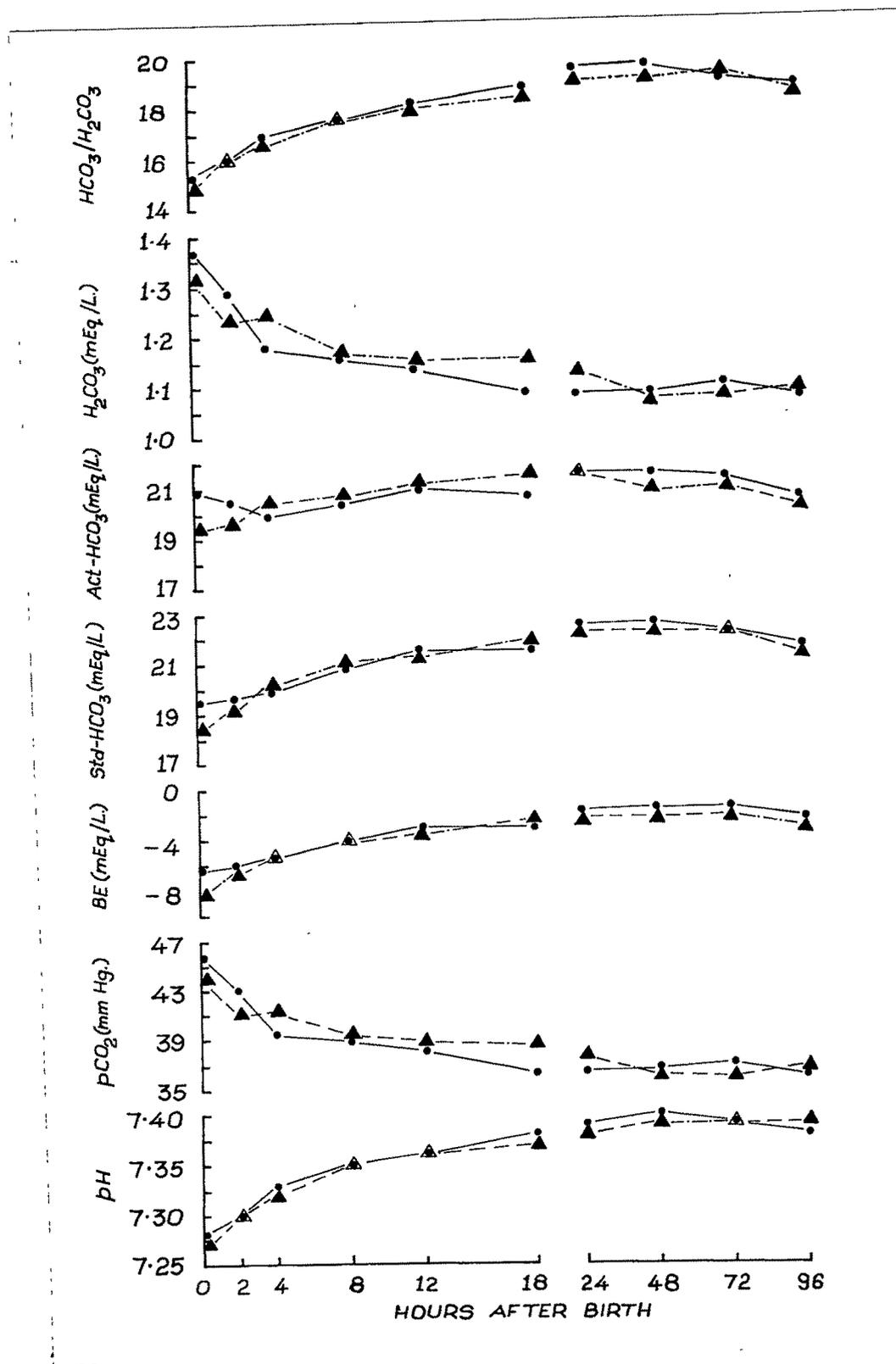


Figure 7.

PLASMA ELECTROLYTE STATUS

RESULTS:

The number of infants studied in this group are 11. Their results are expressed as mean, standard deviation, range and number of determinations in Table 19. Fig.8 demonstrates graphically the behaviour of their mean concentrations.

Plasma Potassium: The mean concentration of plasma potassium at birth is significantly higher ($t = 2.96$, $P < 0.01$) than that of the normal group. The values are within the normal adult range after four hours of age.

Plasma Sodium: The mean concentration of plasma sodium is higher than that of the normal group between two to 18 hours after birth and is lower thereafter. The values remain within the normal adult range throughout the period of observation.

Plasma Chloride: The mean concentration of plasma chloride is found to be higher than that of the normal group except at 18 hours of age.

Plasma Calcium: The pattern of plasma calcium is similar to that of the normal group and the mean concentrations are slightly lower except at two hours after birth.

Plasma Magnesium: The mean concentration of plasma magnesium is lower than that of the normal group except at 72 hours. The values are significantly lower at four hours.

($t = 2.13$, $P < 0.05$) and at eight hours ($t = 2.78$, $P < 0.02$) respectively. The mean concentrations are less than the lower limits of the normal adult range throughout the period of observation.

Plasma Inorganic Phosphorus: The concentration of plasma inorganic phosphorus is lower than that of the normal group from two to 48 hours after birth. Significant decrease ($t = 2.22$, $P < 0.05$) in the concentration is found at 18 hours of age. The highest value of inorganic phosphorus coincides with the lowest concentration of plasma calcium at 72 hours.

Plasma Total Protein: The mean total protein concentration is lower than that of the normal group throughout the whole period but does not show significant difference.

Blood Urea: The mean blood urea concentration is lower upto 48 hours as compared to that of the normal group. However, the difference is significant at four hours ($t = 2.06$, $P < 0.05$) and at eight hours of age ($t = 2.06$, $P < 0.05$). The maximum rise after birth amounts to 120 per cent in this group as compared to 69 per cent in the normal group.

Representative data from the literature on the electrolyte status during the early neonatal period are summarized in Table 19-A.

Jukarainen (1971) carried out a study of plasma calcium, magnesium and phosphorus in 44 infants of low birth weight during the first five days of life. The mean calcium concentration fell from the initial value of 8.8 mg./100 ml. to 7.4 mg./100 ml. at 44 hours after birth. The mean magnesium

showed a rising tendency towards the end of the observation. The maximum value was 2.32 mg./100 ml. at 82 hours which was 14 per cent higher than the lowest value of 2.01 mg./100 ml. at the age of six hours. The mean phosphorus showed a marked initial fall from 7.6 to 6.1 mg./100 ml. immediately after birth. Then the phosphorus concentration rose to a maximum value of 7.6 mg./100 ml. at 44 hours.



DISCUSSION:

The initially higher plasma potassium level shows a continuous fall more pronounced during the first four postnatal hours of life, and the fall is associated with a simultaneous increase in the plasma sodium concentration. These reciprocal changes in the potassium and sodium concentrations could be due to an improvement in the acidosis. The plasma potassium and sodium concentrations do not show much variation thereafter to comment upon. The mean concentrations of plasma magnesium and inorganic phosphorus are lower than those of the normal group upto 24 hours of age. The low trends of the above minerals could be on account of intra-uterine malnutrition which is thought to cause low birth weight. The subsequent rise in the plasma magnesium and phosphorus concentrations seen after 24 hours is due to tissue breakdown. The similar rise observed in the urea concentration (120 per cent) is indicative of a relatively poor renal function and an increased tissue breakdown occurring in these infants as compared to that of the normal group.

TABLE 19.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF HEALTHY LOW BIRTH WEIGHT INFANTS. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.
0	6.00 0.44 8	5.6- 6.9	143.0 5.35 8	134- 150	100.5 5.16 7	95- 108	10.71 0.89 7	9.4- 11.5	1.28 0.35 6	0.9- 1.7	5.06 0.62 8	4.2- 6.1	13.4 2.32 8	10- 18
2	5.42 0.53 11	4.9- 6.3	143.5 4.01 11	134- 150	102.4 7.03 10	93- 116	10.47 0.59 11	9.4- 11.2	1.35 0.39 10	0.9- 2.0	5.01 0.84 11	3.6- 6.6	14.5 3.60 11	9- 22
4	5.11 0.68 11	4.0- 5.9	145.5 6.45 11	128- 152	103.2 7.71 10	93- 118	10.32 0.61 11	9.6- 11.7	1.27 0.32 10	0.8- 1.9	5.06 0.78 11	4.0- 6.5	14.9 3.35 11	10- 20
8	5.01 0.93 11	3.8- 6.3	142.7 5.73 11	132- 150	105.1 6.97 10	93- 118	10.00 0.53 11	9.2- 10.8	1.05 0.26 10	0.7- 1.6	4.74 0.77 11	3.4- 6.3	16.7 3.24 11	10- 21
12	5.00 0.85 11	3.4- 6.2	143.1 5.31 11	134- 156	105.2 6.2 10	95- 116	9.83 0.77 11	8.8- 11.0	1.26 0.34 10	0.8- 1.9	4.71 0.79 11	3.8- 6.3	19.4 4.47 11	13- 29
18	5.01 1.09 11	3.5- 6.8	143.1 2.76 11	138- 148	102.8 5.61 10	93- 114	9.71 0.71 11	8.1- 10.7	1.14 0.39 10	0.6- 2.1	4.80 1.16 11	2.9- 6.5	21.3 4.95 11	15- 31
24	4.90 0.75 11	3.6- 6.2	141.5 3.72 11	134- 146	103.1 6.83 10	93- 118	9.66 0.88 11	8.2- 11.0	1.06 0.29 10	0.6- 1.6	5.5 1.17 11	3.4- 6.8	23.1 8.10 11	15- 44

TABLE 19. (CONTINUED)

Hours after birth	K	Na	Cl	Ca	Mg	Phosphorus	Urea
48	4.96 0.59 11	141.6 5.29 148 11	102.8 4.71 111 10	9.71 0.79 11.0 11	1.35 0.51 2.2 10	6.05 4.2- 1.15 7.7 11	25.3 9- 13.48 57 11
72	4.69 0.84 11	140.0 4.47 136- 150 11	103.7 6.02 114 11	9.42 1.07 7.8- 11.0 11	1.44 0.47 2.4 10	6.13 4.1- 1.03 7.0 11	27.5 8- 20.08 80 11
96	4.39 1.14 10	140.4 5.01 134- 148 10	103.7 3.33 108 10	9.83 0.78 8.2- 11.0 10	1.33 0.48 0.9- 2.4 9	6.08 3.8- 0.87 7.3 10	29.5 10- 29.78 110 10

TABLE 19-A.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF HEALTHY LOW BIRTH WEIGHT INFANTS. (REPRESENTATIVE DATA FROM LITERATURE).

Author (Year) Age	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mEq/L.		Mg mEq/L.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.	SD±	No.
Jukarainen (1971)														
0-2 hr	-	-	-	-	-	-	4.4	-	1.80	-	7.6	-	-	-
							0.6		0.24		2.6			
							21		18		20			
2-8 hr	-	-	-	-	-	-	4.3	-	1.69	-	6.1	-	-	-
							0.7		0.33		2.4			
							24		17		19			
8-16 hr	-	-	-	-	-	-	4.0	-	1.78	-	6.7	-	-	-
							0.8		0.32		2.1			
							35		30		34			
16-24 hr	-	-	-	-	-	-	3.9	-	1.79	-	7.0	-	-	-
							0.7		0.34		3.0			
							41		34		35			
24-32 hr	-	-	-	-	-	-	3.9	-	1.70	-	6.9	-	-	-
							0.8		0.27		2.4			
							37		33		36			
32-40 hr	-	-	-	-	-	-	3.9	-	1.83	-	7.3	-	-	-
							0.7		0.29		1.7			
							37		33		34			
40-48 hr	-	-	-	-	-	-	3.7	-	1.75	-	7.6	-	-	-
							0.6		0.30		2.2			
							36		31		36			

TABLE 19-A. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
48-56 hr	-	-	-	4.0 0.7 37	1.82 0.30 33	6.8 1.8 36	-
56-64 hr	-	-	-	3.7 0.8 34	1.80 0.31 30	7.0 2.0 33	-
64-76 hr	-	-	-	4.0 0.6 37	1.85 0.28 29	6.5 2.0 31	-
76-88 hr	-	-	-	4.0 0.6 36	1.93 0.34 28	6.4 1.9 31	-
88-100 hr	-	-	-	4.0 0.7 30	1.86 0.29 23	6.3 1.9 26	-
100-128 hr	-	-	-	4.1 0.7 30	1.88 0.30 26	5.8 1.7 26	-

FIGURE 8.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM,
INORGANIC PHOSPHORUS, TOTAL PROTEIN AND BLOOD UREA
CONCENTRATIONS (MEAN) OF FULL-TERM NORMAL INFANTS
(●———●) AND HEALTHY LOW BIRTH WEIGHT INFANTS
(▲- - - -▲).

The scale is reduced after 18 hours.

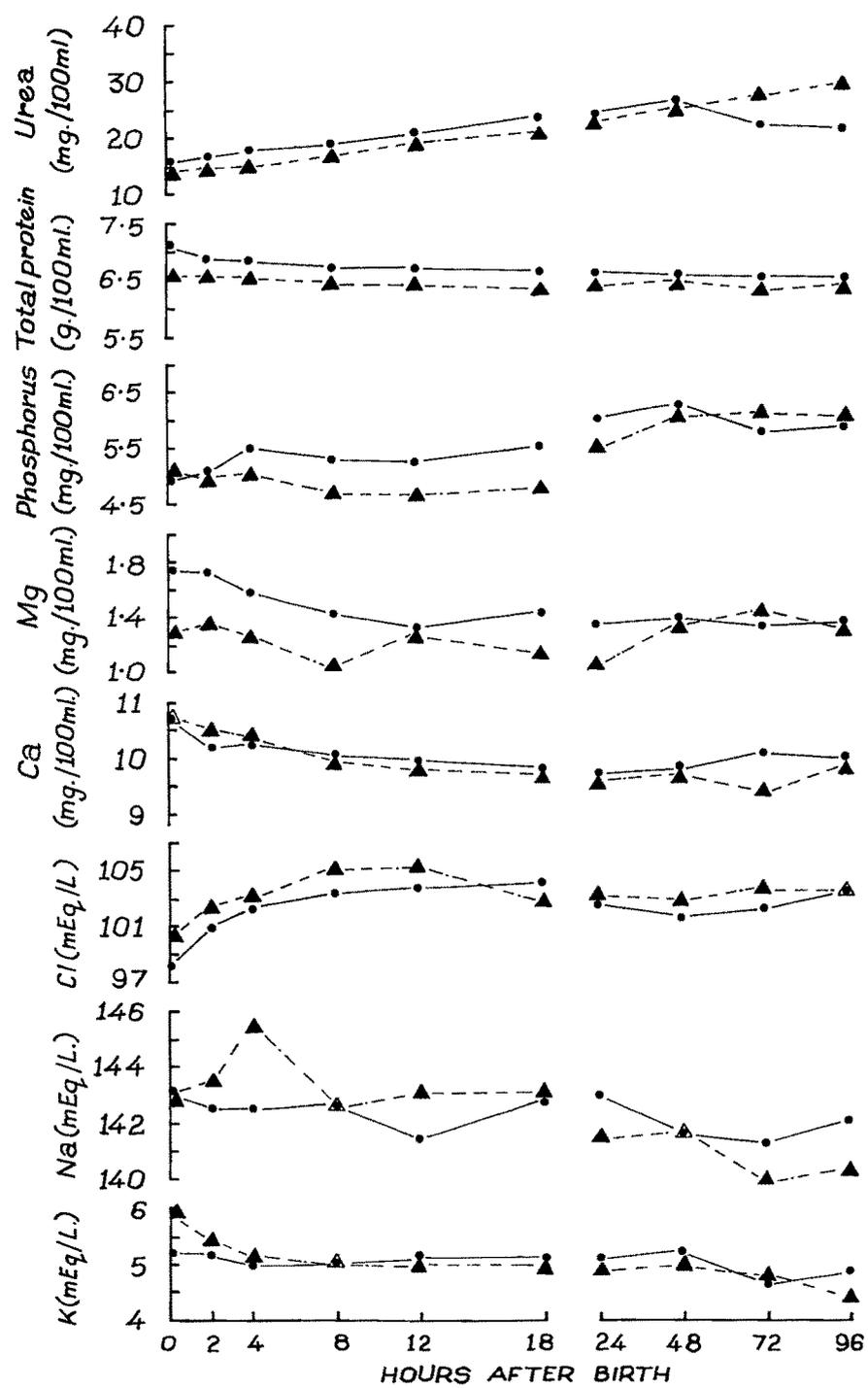


Figure 8.

PLASMA PROTEIN FRACTIONS

RESULTS:

In the present group, the electrophoretic separation of plasma protein is carried out for a period of 96 hours after birth in 11 infants and their mothers at delivery. The results are expressed as mean, standard deviation, range and number of determinations in Table 20.

Plasma Albumin: The mean concentration of plasma albumin is below the normal adult range and is significantly lower than that of the normal group at zero, two, four, eight, 12, 18 and 24 hours after birth ($P < 0.01$, < 0.01 , < 0.02 , < 0.02 , < 0.01 , < 0.01 and < 0.02 respectively). However, the concentration does not show much variation during the whole period. The maternal plasma albumin concentration is much lower than that of the infant at birth.

Plasma Alpha₁, Alpha₂ and Beta: The mean concentration of these globulin fractions are higher than those of the normal group. A significant increase ($t = 2.04$, $P < 0.05$) in the alpha₁ fraction is found at 24 hours of age.

Plasma Gamma Globulin: The mean concentration of plasma gamma globulin is 1.55 g./100 ml. at birth which gradually falls to 1.28 g./100 ml. at 96 hours. The fall is 17 per cent.

The concentrations of the infants' globulin fractions are much lower than those of the mother.

DISCUSSION:

The significant lower concentration of albumin at birth and thereafter as compared to that of the normal could be due to a diminished synthesis of albumin probably on account of an intra-uterine growth retardation resulting in functional immaturity of the liver. No explanation could be put forth for a significant rise in alpha₁ concentration at 24 hours as compared to that in the normal group. The 23 per cent fall in beta globulin concentration occurring at 18 hours could be on account of the fluid shift. The concentrations of gamma globulins of this group at birth and subsequently are comparable to those of the normal group. This reflects an adequacy of the placental transfer of maternal gamma globulins in the low birth weight neonate delivered at term in contrast to the pre-term (premature) infant.

TABLE 20*

PLASMA TOTAL PROTEIN AND PROTEIN FRACTIONS (g./100 ml.) OF HEALTHY LOW BIRTH WEIGHT INFANTS AND THEIR MOTHERS AT DELIVERY. (PRESENT SERIES)

Hours after birth	T. Protein			Globulin									
	Mean	Range	SD± No.	Albumin		Alpha ₁		Alpha ₂		Beta		Gamma	
	Mean	Range	SD± No.	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Mothers at delivery	6.56	5.50-7.50	0.54 11	2.56	2.25-3.15	0.33	0.20-0.45	0.73	0.53-0.96	1.14	0.91-1.70	1.80	1.21-2.24
				(39.0)		(5.0)		(11.1)		(17.4)		(27.5)	
				0.28		0.09		0.09		0.23		0.41	
				11		11		11		11		11	
0	6.55	6.00-7.50	0.45 8	3.71	3.30-4.40	0.21	0.12-0.30	0.39	0.28-0.55	0.69	0.56-0.85	1.55	1.36-1.89
				(56.7)		(3.2)		(5.9)		(10.4)		(23.8)	
				0.40		0.07		0.08		0.12		0.19	
				8		8		8		8		8	
2	6.55	6.00-7.30	0.41 11	3.64	3.05-4.70	0.20	0.15-0.35	0.44	0.25-0.56	0.72	0.42-1.28	1.55	1.21-1.83
				(55.6)		(3.1)		(6.7)		(11.0)		(23.6)	
				0.47		0.06		0.10		0.23		0.20	
				11		11		11		11		11	
4	6.53	5.85-7.40	0.42 11	3.69	2.85-4.40	0.20	0.16-0.26	0.43	0.32-0.53	0.64	0.43-1.00	1.57	1.14-2.14
				(56.6)		(3.0)		(6.5)		(9.9)		(24.0)	
				0.42		0.03		0.07		0.15		0.27	
				11		11		11		11		11	
8	6.43	5.75-7.45	0.50 11	3.69	3.20-4.43	0.21	0.12-0.28	0.42	0.24-0.65	0.62	0.36-0.86	1.48	1.14-1.89
				(57.5)		(3.2)		(6.6)		(9.7)		(23.0)	
				0.40		0.05		0.11		0.14		0.28	
				11		11		11		11		11	
12	6.42	5.85-7.20	0.45 11	3.72	3.30-4.35	0.19	0.12-0.31	0.39	0.30-0.51	0.62	0.43-1.10	1.50	1.17-1.90
				(58.1)		(3.0)		(6.0)		(9.7)		(23.2)	
				0.31		0.05		0.07		0.18		0.22	
				11		11		11		11		11	

TABLE 20. (CONTINUED)

Hours after birth	T. Protein	Albumin	Globulin						
			Alpha ₁	Alpha ₂	Beta	Gamma			
18	6.36 0.57 11	3.76 (59.2) 0.36 11	0.21 (3.2) 0.07 11	0.37 (5.8) 0.08 11	0.24- 0.51	0.53 (8.3) 0.14 11	0.28- 0.73	1.49 (23.5) 0.25 11	1.14- 1.95
24	6.38 0.42 11	3.68 (57.8) 0.37 11	0.24 (3.7) 0.06 11	0.39 (6.1) 0.12 11	0.22- 0.64	0.60 (9.4) 0.16 11	0.40- 0.90	1.47 (23.0) 0.30 11	1.05- 1.87
48	6.46 0.45 11	3.92 (61.0) 0.42 11	0.23 (3.5) 0.08 11	0.39 (6.1) 0.09 11	0.24- 0.51	0.57 (8.7) 0.14 11	0.34- 0.71	1.35 (20.7) 0.39 11	0.82- 1.86
72	6.31 0.48 11	3.73 (59.0) 0.69 11	0.25 (4.1) 0.06 11	0.43 (6.8) 0.09 11	0.32- 0.62	0.61 (9.6) 0.14 11	0.43- 0.91	1.29 (20.5) 0.26 11	0.98- 1.84
96	6.30 0.59 10	3.75 (59.4) 0.46 10	0.22 (3.5) 0.04 10	0.44 (7.0) 0.09 10	0.35- 0.63	0.61 (9.6) 0.11 10	0.41- 0.77	1.28 (20.5) 0.28 10	0.80- 1.64

Figures in the parenthesis indicate percentage of total protein.

INFANTS WITH MATERNAL TOXAEMIA

- (i) Infants delivered by difficult obstetric procedures.
- (ii) Low birth weight infants.

ACID-BASE STATUS

Toxaemias of pregnancy claim a heavy toll of foetal lives and contributes in large measure to maternal morbidity and mortality.

RESULTS (i)

This group includes three infants. The results of acid-base parameters are expressed as mean, range and number in Table 21. Fig.9 demonstrates their mean changes graphically.

pH: The mean pH value of 7.14 at birth is very much lower than that of the similar group without maternal toxaemia. The pH attains the lower limits of the normal adult range at 18 hours and remains between 7.40 to 7.42 from 24 to 72 hours of age.

pCO₂: The mean concentration is found much higher at birth and lower between two to 18 hours than that of similar group without maternal toxaemia.

BE, Standard HCO₃ and Actual HCO₃: The mean concentrations of these parameters are lower than those of the similar group without maternal toxaemia from birth to 18 hours and little higher afterwards.

H₂CO₃: It follows a trend similar to that of pCO₂.

HCO₃/H₂CO₃: The ratio is 11.2 at birth, which is about 56 per cent of the normal. It rises gradually and attains the normal value at 24 hours of age.

RESULTS (ii):

The number of infants studied in this group is four. The results are expressed as mean, range and number of determinations in Table 21-A. Their mean concentrations are graphically demonstrated in Fig. 10.

pH: The mean pH value at birth is 7.27 and attains the lower limits of the normal adult range at 12 hours. The values are lower than those of the corresponding group without maternal toxæmia at two, four, eight, 72 and 96 hours after birth.

pCO₂: The mean pCO₂ values are lower than those of the similar group without maternal toxæmia upto 72 hours, more marked from eight to 24 hours.

BE, Standard HCO₃ and Actual HCO₃: The mean concentrations of these parameters show a marked decrease than those of the corresponding group without maternal toxæmia upto 72 hours of age.

H₂CO₃: It shows a trend similar to that of pCO₂.

HCO₃/H₂CO₃: The behaviour is similar to that of the corresponding group without toxæmia.

DISCUSSION:

Infants in both the groups show non-respiratory acidosis from birth onwards till 12 to 18 hours except for the initial respiratory acidosis seen in the infants delivered by difficult obstetric procedures. This non-respiratory acidosis could be on account of a more severe type of hypoxia causing an increased glycolysis leading to a greater degree of lactacidosis. It is possible that such a situation could occur due to placental insufficiency in maternal toxæmia (Holman and Lipsitz, 1966). The compensatory hyperventilation seems to be responsible for the respiratory basosis observed during the period of 72 hours in the low birth weight group. The relatively higher degree of compensatory hyperventilation found in this group is on account of poor sensibilization of the respiratory centre which is also observed in the corresponding non-toxæmic group.

TABLE 21.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE, CARBONIC ACID AND BICARBONATE/CARBONIC ACID CONCENTRATIONS OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES WITH MATERNAL TOXAEMIA. (PRESENT SERIES)

Hours after birth	pH		pCO ₂ mm.Hg		BE mEq/L.		Std.HCO ₃ mEq/L. Plasma		Actual HCO ₃ mEq/L. Plasma		H ₂ CO ₃ mEq/L. Plasma		HCO ₃ /H ₂ CO ₃	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	No.		No.		No.		No.	No.		No.		No.	No.	
0	7.14	7.06-	52.3	50.0-	-13.7	-18.5	15.1	12.2-	17.6	14.3-	1.58	1.50-	11.2	9.0-
	3	7.29	3	57.0	3	-4.5	3	20.7	3	23.0	3	1.71	3	15.3
2	7.21	7.10-	40.0	30.0-	-12.9	-20.3	15.8	12.0-	15.8	12.0-	1.20	0.90-	13.0	10.0-
	3	7.29	3	50.0	3	-4.5	3	20.7	3	23.0	3	1.50	3	15.3
4	7.29	7.20-	34.0	30.0-	-8.8	-15.3	17.5	13.7-	16.1	12.0-	1.02	0.90-	15.6	12.4-
	3	7.35	3	40.0	3	-3.8	3	21.3	3	21.3	3	1.20	3	17.7
8	7.31	7.25-	35.0	30.0-	-7.9	-13.3	18.5	15.0-	17.1	12.6-	1.08	0.90-	16.2	14.4-
	3	7.36	3	37.5	3	-3.8	3	21.2	3	20.3	3	1.20	3	18.4
12	7.30	7.28-	34.7	33.5-	-8.6	-10.5	18.2	17.0-	16.6	15.2-	1.04	1.00-	16.0	15.0-
	3	7.35	3	36.0	3	-5.8	3	19.8	3	18.3	3	1.08	3	17.7
18	7.37	7.36-	35.3	27.5-	-3.8	-6.0	21.1	19.7-	19.6	15.7-	1.06	0.83-	18.5	18.2-
	3	7.38	3	41.0	3	-2.2	3	22.2	3	22.4	3	1.23	3	18.9
24	7.40	7.37-	35.7	32.0-	-1.7	-3.0	22.0	21.7	21.3	20.5-	1.07	0.96-	20.0	18.7-
	3	7.44	3	37.5	3	-1.0	3	23.2	3	22.5	3	1.12	3	21.4

TABLE 21. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.41 7.39- 7.44	35.5 33.0- 37.5	-1.3 -2.5 -0.5	23.0 22.2- 23.5	21.5 20.8- 22.3	1.06 0.99- 1.12	20.3 19.2- 21.7
72	7.42 7.40- 7.45	35.5 33.0- 37.5	-0.8 -1.8 +0.3	23.2 22.5- 24.0	22.1 22.0 22.3	1.06 0.99- 1.12	20.9 20.0- 22.2
96	7.39 7.38- 7.40	36.2 35.0- 37.5	-1.8 -3.0 -1.0	22.0 21.5- 22.7	21.7 20.5- 22.5	1.08 1.05- 1.12	20.0 19.0- 20.9

FIGURE 9.

BLOOD pH, pCO_2 , BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE, CARBONIC ACID AND BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES (▼— — — ▼) AND INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES WITH MATERNAL TOXAEMIA (▽— — — ▽).

The scale is reduced after 18 hours.

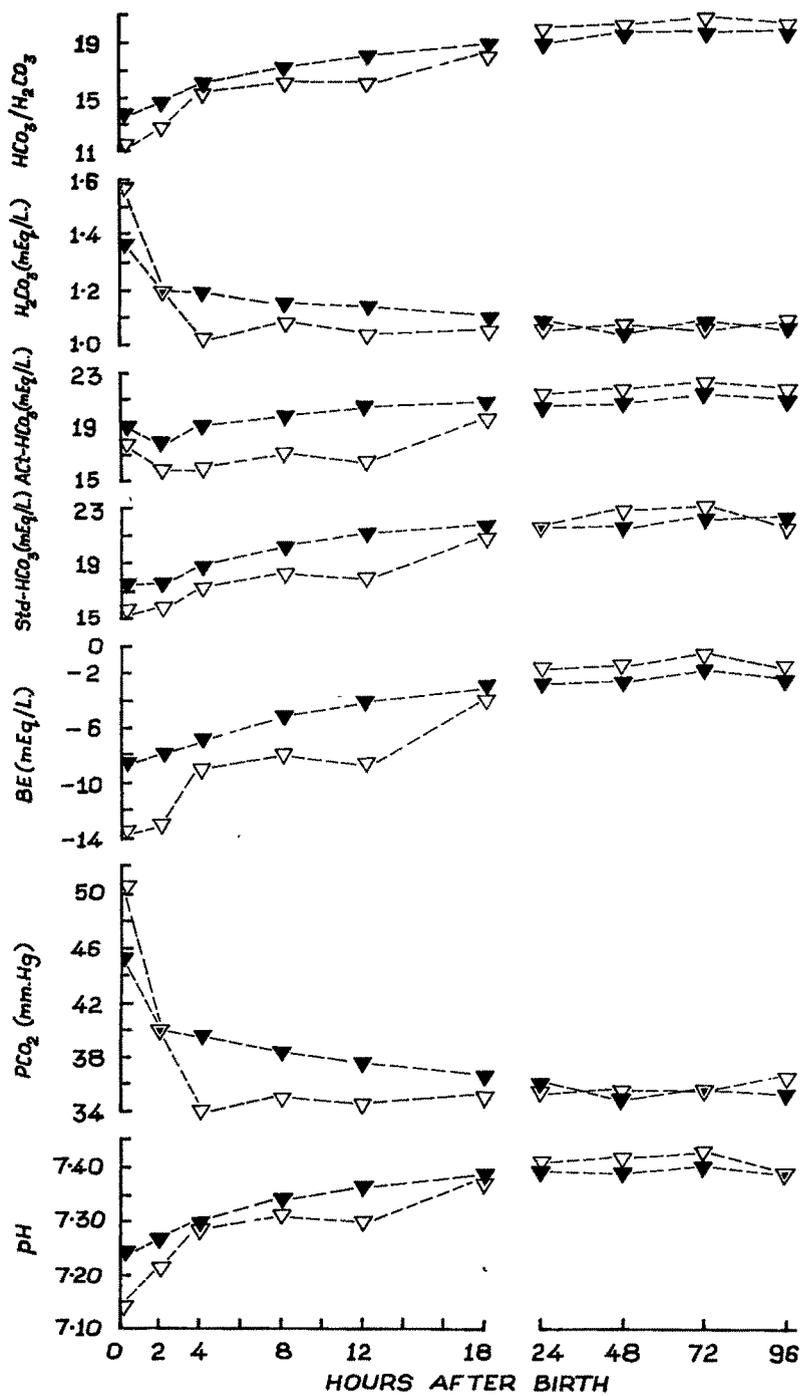


Figure 9.

TABLE 21-A.

BLOOD pH, pCO₂, BASE EXCESS, STANDARD BICARBONATE, ACTUAL BICARBONATE, CARBONIC ACID AND BICARBONATE/CARBONIC ACID CONCENTRATIONS OF LOW BIRTH WEIGHT INFANTS WITH MATERNAL TOXAEMIA.
(PRESENT SERIES)

Hours after birth	pH		pCO ₂ mm.Hg		BE mEq/L.		Std. HCO ₃ mEq/L. Plasma		Actual HCO ₃ mEq/L. Plasma		H ₂ CO ₃ mEq/L. Plasma		HCO ₃ /H ₂ CO ₃	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
0	7.28	7.26-7.31	39.7	35.5-44.0	-7.7	-10.7-4.8	18.4	16.3-20.5	18.3	15.3-21.3	1.19	1.06-1.32	15.2	14.4-16.1
2	7.25	7.15-7.33	39.1	25.5-51.0	-10.2	-20.8-4.5	17.3	11.7-20.7	17.1	8.6-23.2	1.17	0.76-1.53	14.3	11.3-16.8
4	7.29	7.23-7.34	39.2	25.5-48.0	-7.7	-16.0-4.0	18.6	13.4-21.0	18.6	10.3-22.7	1.13	0.60-1.44	15.6	13.5-17.4
8	7.34	7.29-7.36	33.2	20.0-44.0	-6.8	-14.3-2.3	19.4	14.7-22.2	17.5	9.3-23.5	1.04	0.79-1.32	17.2	15.5-18.0
12	7.36	7.33-7.38	33.1	26.5-37.5	-5.6	-8.6-2.3	19.9	18.0-22.2	17.9	14.0-21.3	0.97	0.69-1.12	18.0	17.0-19.0
18	7.37	7.36-7.38	32.4	23.0-40.0	-4.8	-8.0-1.5	20.5	18.5-22.7	18.2	13.0-22.7	0.97	0.69-1.20	19.1	18.8-20.0
24	7.39	7.37-7.42	32.5	23.0-40.0	-3.1	-8.0-1.0	21.3	18.5-23.3	19.3	13.0-23.3	1.01	0.85-1.20	19.7	18.8-20.8

TABLE 21-A. (CONTINUED)

Hours after birth	pH	pCO ₂	BE	Std. HCO ₃	Actual HCO ₃	H ₂ CO ₃	HCO ₃ /H ₂ CO ₃
48	7.39	33.6	-3.0	21.8	20.0	1.05	19.8
4	7.33-	28.5-	-9.2	17.7-	14.5-	0.90-	17.0-
	7.43	40.0	-0.3	24.0	24.0	1.20	21.3
72	7.36	33.7	-4.8	20.4	18.6	1.01	18.2
4	7.35-	32.0-	-5.8	19.8-	18.1-	0.96-	17.1-
	7.38	35.0	-4.0	21.3	19.5	1.05	19.2
96	7.36	37.6	-2.4	22.2	21.4	1.12	18.9
4	7.38-	33.0-	-6.8	19.3-	17.2-	0.96-	17.3-
	7.40	44.0	+2.0	25.5	26.3	1.32	20.0

FIGURE 10.

BLOOD pH, pCO_2 , BASE EXCESS, STANDARD BICARBONATE,
ACTUAL BICARBONATE, CARBONIC ACID AND
BICARBONATE/CARBONIC ACID CONCENTRATIONS (MEAN) OF
HEALTHY LOW BIRTH WEIGHT INFANTS (\blacktriangle —·—·—· \blacktriangle) AND
LOW BIRTH WEIGHT INFANTS WITH MATERNAL TOXAEMIA
(\triangle —·—·—· \triangle).

The scale is reduced after 18 hours.

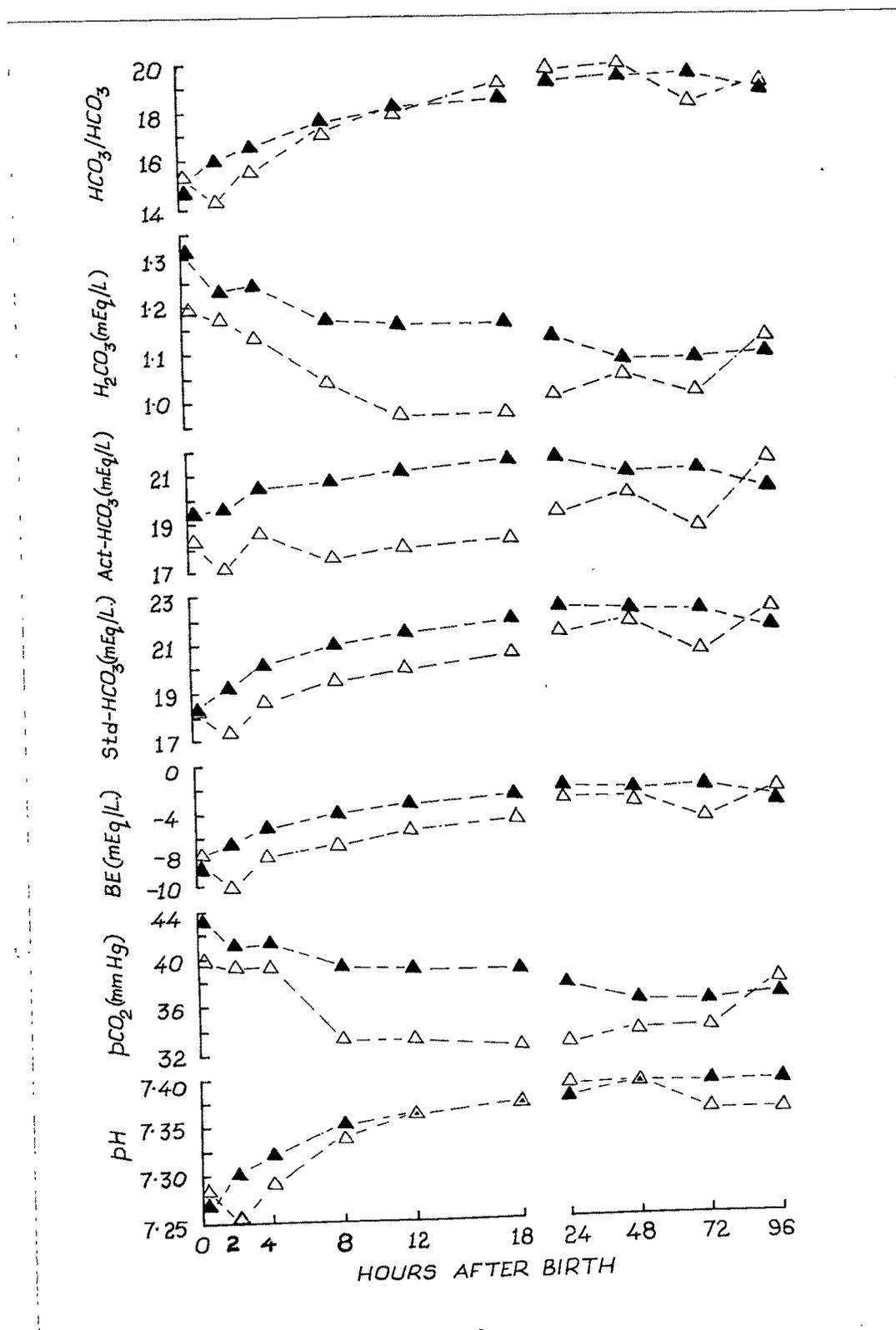


Figure 10.

PLASMA ELECTROLYTE STATUS

RESULTS (i):

The electrolyte parameters are studied in three infants during 96 hours after birth. The results are expressed as mean, range and number in Table 22. The behaviour of the mean concentrations of these parameters is shown graphically in Fig.11.

Plasma Potassium: The mean concentrations of plasma potassium are higher than that of the normal adult range upto 72 hours and do not show much deviation from those of the similar group without maternal toxæmia. A gradual fall to the extent of 25 per cent is seen during the period of study.

Plasma Sodium and Chloride: The mean concentrations of plasma sodium are lower except at 24 hour, while the mean concentrations of plasma chloride are higher except upto two hours as compared to the similar group without maternal toxæmia.

Plasma Calcium: The trend of mean concentration of plasma calcium is similar to that of the corresponding group without maternal toxæmia.

Plasma Magnesium: The level of mean concentration of plasma magnesium is found highest in this group as compared to other groups in the present study.

Plasma Inorganic Phosphorus: The mean concentration of plasma inorganic phosphorus is much lower throughout the

period of observation than that of the similar group without maternal toxæmia. The minimum concentration of 4.16 mg./100 ml. at four hour shows a rise of 25 per cent at 48 hours.

Plasma Total Protein: The mean total protein concentration is lower than that of the similar group without maternal toxæmia.

Blood Urea: The mean blood urea concentration is higher than that of the similar group without maternal toxæmia upto 48 hours. The mean value of 43.0 mg./100 ml. at 48 hours indicates a rise of 79 per cent as compared to the concentration of 24.0 mg./100 ml. at birth. The magnitude of rise observed in the blood urea concentration in both the groups is nearly same.

RESULTS (ii):

The number of infants studied in this group is four. The results are expressed as mean, range and number in Table 22-A. Fig.12 demonstrates the behaviour of their mean concentrations graphically.

Plasma Potassium: The mean concentrations of plasma potassium are higher than that of the normal adult range upto 12 hours and do not show much variation from those of the corresponding group without maternal toxæmia. A gradual fall to the extent of 24 per cent is seen during the study.

Plasma Sodium: The mean concentrations of plasma sodium are lower than those of the similar group except at birth, 48 and 72 hours.

Plasma Chloride: The mean concentrations are much lower than those of the corresponding group without maternal toxæmia throughout the period of observation.

Plasma Calcium: The mean concentration of plasma calcium is lower than the corresponding group without maternal toxæmia upto 48 hours, but the values are within the normal adult range. The fall in plasma calcium is not marked in this group except at two hours.

Plasma Magnesium: The level of plasma magnesium is much higher throughout the period of 96 hours as compared to the similar group without maternal toxæmia.

Plasma Inorganic Phosphorus: The mean concentration of inorganic phosphorus is 3.55 mg./100 ml. at birth and shows a rise of 74 per cent at 48 hours as compared to 25 per cent seen in the corresponding group without maternal toxæmia.

Plasma Total Protein: The mean total protein values are lower than those of the similar group without maternal toxæmia.

Blood Urea: The mean concentrations of blood urea are higher, the change being more marked after 24 hours as compared to that of the corresponding group without maternal toxæmia. The mean value of 12.0 mg./100 ml. at birth shows a rise of 314 per cent at 96 hours.

Representative data from the literature on the electrolyte status during the early neonatal period are summarized in Table 22-B.

Jukarainen (1971) studied the behaviour of plasma calcium, magnesium and phosphorus in 10 low birth weight infants of toxæmic mothers during the first five days of life. Initially, plasma calcium remained at a high level of 9.4 mg./100 ml. upto 12 hours, subsequently fell at 60 hours to the lowest value of 7.8 mg./100 ml. The fall of calcium was not very marked in this group. The mean concentration of plasma magnesium was lower than that of the corresponding group without maternal toxæmia (Table 19-A). Plasma phosphorus showed a marked fall initially at two hours, thereafter the level rose to the highest value at 36 hours with an increase of 46 per cent.

DISCUSSION:

The plasma potassium values in both the groups do not show much deviation from those of the corresponding non-toxaemic groups. During delivery, toxaemic mothers are usually given parenteral fluids which are passively transferred to the infant. This would lead to haemodilution which may account for low plasma sodium concentrations seen in the infants having maternal toxaemia (Holman and Lipsitz, 1966). The low plasma chloride concentrations seen in the low birth weight group also appear to be related to the haemodilution. The initial sharp fall seen in plasma calcium concentration in the low birth weight infants with maternal toxaemia may be due to a corresponding fall in the pH (acidosis). Higher plasma magnesium concentrations seen in the infants of the present group could be due to either administration of magnesium therapy to the mothers of these infants or a greater degree of acidosis and tissue breakdown. Magnesium therapy was given only to the mother of infant T 3 before delivery. However, the other infants also showed higher concentrations of magnesium. Thus increased tissue breakdown and acidosis appear to be responsible for this change. Increased tissue breakdown would also account for the higher concentrations of blood urea.

The low total protein concentration of the infants of the toxaemic groups could probably be due to a reduction in the transfer of gamma globulin fraction occurring as a result of placental insufficiency.

TABLE 22.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF INFANTS DELIVERED BY DIFFICULT OBSTETRIC PROCEDURES WITH MATERNAL TOXAEMIA. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	No.		No.		No.		No.	No.		No.		No.	No.	
0	5.96	5.1-6.7	141.3	136-152	96.0	93-102	10.40	9.6-11.0	2.70	1.5-4.5	5.53	4.8-6.0	24.0	17-28
2	5.80	5.3-6.6	140.0	136-144	96.0	93-102	10.00	9.2-10.8	2.43	1.2-4.1	4.86	4.4-5.4	25.0	14-38
4	5.56	5.0-6.2	140.0	136-146	99.3	95-105	9.83	8.9-10.4	2.70	1.5-4.1	4.16	3.1-4.8	28.3	17-35
8	5.50	5.2-5.8	139.3	138-142	101.0	95-110	9.83	8.9-10.4	2.40	1.5-3.6	4.33	4.2-4.6	26.6	18-35
12	5.10	4.6-5.4	141.3	140-142	99.7	95-108	9.36	8.1-10.6	2.43	1.1-4.2	4.30	3.5-4.7	33.7	20-45
18	5.26	4.3-6.1	140.0	142-148	98.0	91-105	10.03	8.5-11.6	2.46	1.2-4.1	4.33	3.5-5.8	36.3	27-40
24	5.26	5.0-5.6	142.0	140-144	100.7	96-108	9.56	8.1-10.8	2.46	1.2-4.1	4.93	3.6-5.8	41.7	26-50

TABLE 22. (CONTINUED)

Hours after birth	K	Na	Cl	Ca	Mg	Phosphorus	Urea				
48	5.10 3	140.7 3	98.7 3	9.36 3	8.5- 10.0	1.43 3	1.0- 1.7	5.56 3	4.6- 6.4	43.0 3	27- 53
72	5.03 3	140.0 3	102.0 3	10.23 3	8.7- 11.2	2.50 3	1.9- 3.3	5.46 3	5.4- 5.8	38.7 3	20- 50
96	4.46 3	142.7 3	104.7 3	10.20 3	9.2- 11.0	1.96 3	1.6- 2.5	5.46 3	4.2- 6.4	26.6 3	14- 45

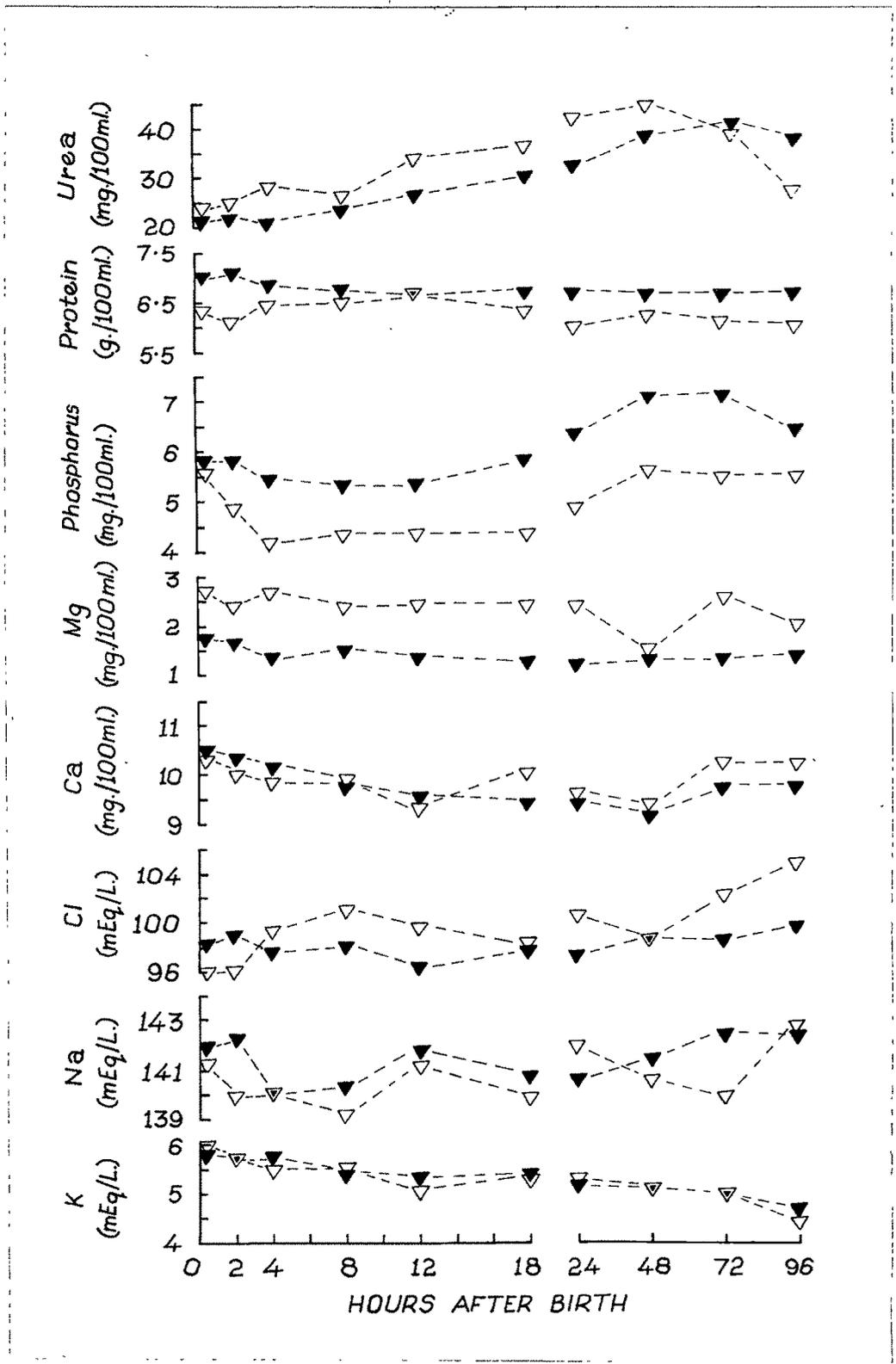


Figure 11.

TABLE 22-A.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF LOW BIRTH WEIGHT INFANTS WITH MATERNAL TOXAEMIA. (PRESENT SERIES)

Hours after birth	K mEq/L.		Na mEq/L.		Cl mEq/L.		Ca mg./100 ml.		Mg mg./100 ml.		Phosphorus mg./100 ml.		Urea mg./100 ml.	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	No.		No.		No.		No.	No.		No.		No.	No.	
0	5.60	5.5-5.7	145.0	142-148	97.5	95-100	10.70	9.1-12.3	2.20	2.0-2.4	3.55	3.2-3.9	12.0	9-15
2	5.27	4.4-6.2	143.5	128-156	95.0	82-102	9.36	8.1-10.5	2.40	2.0-2.8	3.90	3.7-4.2	21.0	3-62
4	5.15	4.0-6.5	139.0	124-152	96.5	82-104	9.57	8.3-10.4	1.97	1.4-2.5	3.60	3.1-3.9	22.7	3-67
8	5.15	4.4-6.0	140.5	124-154	94.7	77-104	9.42	8.1-10.2	2.0	1.8-2.1	4.43	3.8-5.3	26.1	7-68
12	5.17	4.2-6.4	139.0	124-146	95.5	75-103	9.30	7.9-10.2	2.07	1.6-2.3	5.50	5.0-6.4	29.1	13-68
18	5.00	4.0-5.9	140.5	124-150	95.0	73-105	9.47	8.1-11.1	1.90	1.5-2.2	5.63	4.6-6.6	29.7	13-74
24	4.92	3.4-6.2	141.5	128-148	96.5	73-107	9.32	8.7-10.3	1.90	1.5-2.2	6.16	5.7-7.0	33.0	13-80

TABLE 22-A. (CONTINUED)

Hours after birth	K	Na	Cl	Ca	Mg	Phosphorus	Urea				
48	4.76 3	143.0 4	99.5 4	9.55 4	8.7- 10.6	2.10 4	1.7- 2.7	6.20 3	5.8- 6.9	44.5 4	14- 98
72	4.81 3	141.5 4	98.5 4	9.65 4	8.7- 10.5	1.95 4	1.3- 2.8	5.96 3	4.8- 6.9	48.5 4	10- 111
96	4.26 3	140.5 4	98.2 4	9.92 4	9.1- 10.5	1.87 4	1.2- 2.5	5.30 3	4.1- 6.5	49.7 4	6- 108

TABLE 22-B.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM, INORGANIC PHOSPHORUS AND BLOOD UREA CONCENTRATIONS OF LOW BIRTH WEIGHT INFANTS WITH MATERNAL TOXAEMIA.
(REPRESENTATIVE DATA FROM LITERATURE)

Author (Year) Age	K		Na		Cl		Ca		Mg		Phosphorus		Urea	
	Mean	Range	Mean	Range	Mean	Range								
	SD±	No.	SD±	No.	SD±	No.								
Jukerainen (1971)														
0-2 hr	-	-	-	-	-	-	4.7	-	1.77	-	6.7	-	-	-
							0.5		0.20		3.1			
							7		7		7			
2-8 hr	-	-	-	-	-	-	4.7	-	1.58	-	4.9	-	-	-
							0.4		0.21		0.7			
							5		5		5			
8-16 hr	-	-	-	-	-	-	4.7	-	1.57	-	5.7	-	-	-
							0.3		0.22		2.3			
							6		6		6			
16-24 hr	-	-	-	-	-	-	4.4	-	1.64	-	6.0	-	-	-
							0.6		0.25		2.9			
							9		9		9			
24-32 hr	-	-	-	-	-	-	4.2	-	1.45	-	6.3	-	-	-
							0.5		0.18		2.0			
							9		9		9			
32-40 hr	-	-	-	-	-	-	4.1	-	1.73	-	7.2	-	-	-
							0.6		0.26		2.0			
							8		8		8			
40-48 hr	-	-	-	-	-	-	4.1	-	1.50	-	6.9	-	-	-
							0.5		0.26		1.9			
							7		7		7			

TABLE 22-B. (CONTINUED)

Author (Year) Age	K	Na	Cl	Ca	Mg	Phosphorus	Urea
48-56 hr	-	-	-	4.1 0.7 8	1.68 0.41 8	6.3 1.3 8	-
56-64 hr	-	-	-	3.9 0.6 8	1.60 0.23 8	6.1 1.5 8	-
64-76 hr	-	-	-	4.2 0.5 8	1.69 0.25 8	5.6 1.4 8	-
76-88 hr	-	-	-	4.2 0.5 6	1.84 0.40 6	5.7 1.3 6	-
88-100 hr	-	-	-	4.4 0.4 6	1.68 0.34 6	6.0 1.5 6	-
100-128 hr	-	-	-	4.5 0.4 7	1.76 0.35 7	5.9 1.0 7	-

FIGURE 12.

PLASMA POTASSIUM, SODIUM, CHLORIDE, CALCIUM, MAGNESIUM,
INORGANIC PHOSPHORUS, TOTAL PROTEIN AND BLOOD UREA
CONCENTRATIONS (MEAN) OF HEALTHY LOW BIRTH WEIGHT
INFANTS (▲---▲) AND LOW BIRTH WEIGHT INFANTS
WITH MATERNAL TOXAEMIA (△---△).

The scale is reduced after 18 hours.

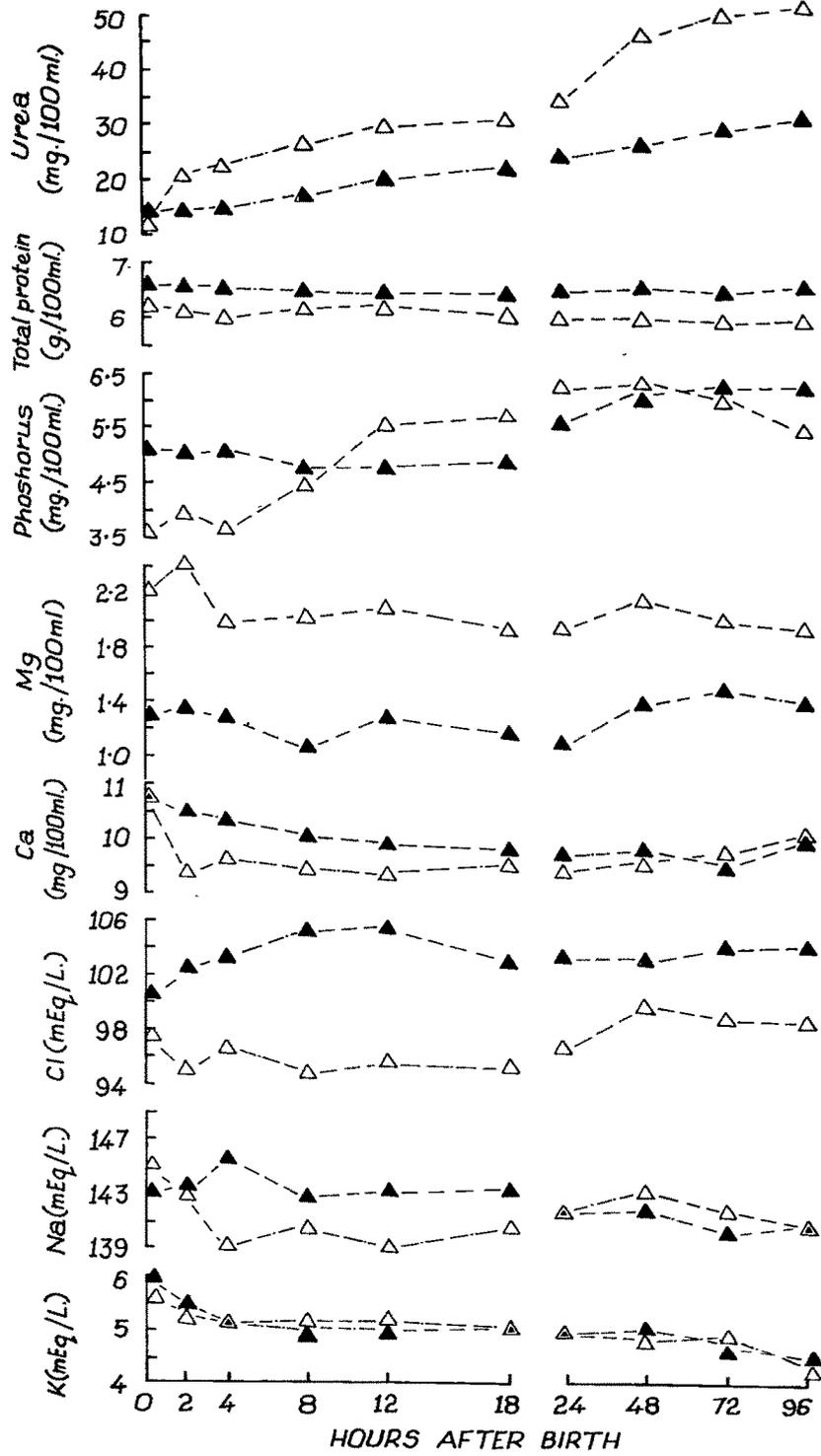


Figure 12.

GENERAL COMMENTS

As birth involves certain amount of trauma, a baby is born with some degree of hypoxia. Hypoxia leads to an increase in the H-ion concentration and $p\text{CO}_2$ while a decrease is seen in BE and standard HCO_3 concentrations. Adaptation and recovery mechanisms of the neonate are much faster as compared to an older child or an adult so that the biochemical picture is normalized earlier. It depends on the mode of delivery and intra-uterine conditions during the foetal age. All the infants in the present study show a disturbed acid-base balance at birth. Most of them exhibit a non-respiratory acidosis, however, a few infants show either combined or respiratory acidosis. The respiratory component is removed much earlier in the normal infants, while the infants delivered by difficult obstetric procedures and premature infants take somewhat longer time to attain the normal respiratory balance. The acidosis at birth calls forth the compensatory mechanism of hyperventilation which is seen in all the groups of infants except in the premature. It appears from the results of the present series that the sensitivity of the respiratory centre to stimulation is poor in the premature infants. Hence, the compensatory hyperventilation seems to be deficient which is depicted in the form of a lowered incidence of respiratory basosis in this group. The compensatory hyperventilatory response is adequate in the normal infants to

bring about acid-base homeostasis in the maximum number of infants (63 per cent) by 24 hours. However, the hyperventilation continues further in the normal neonate because of the slow sensibilization of the respiratory centre resulting in the development of respiratory basosis in about 25 per cent of the infants. The progress of acid-base status in the infants of difficult delivery and low birth weight groups shows a divergence from the pattern found in the normal infants. A relatively much poor sensibilization of the respiratory centre is seen in these infants thus resulting in an increased incidence of respiratory basosis, more marked in the low birth weight infants. The incidence of respiratory basosis is further aggravated in the infants of toxæmic mothers. A greater degree of non-respiratory acidosis seen in the premature infants after 24 hours is due to an increased tissue catabolism and diminished renal function.

The severity of the hypoxia and acidosis, the degree of stress and trauma of birth and the adrenocortical activity in response to stimulation are the major factors which seem to influence the behaviour of the electrolyte status during the neonatal period. The most striking changes are observed in the plasma potassium, sodium, chloride, magnesium, inorganic phosphorus and urea concentrations. The normal neonate undergoes some degree of hypoxia at birth as evidenced by the changes in the acid-base status. This results in an initially higher plasma potassium concentration during the early

post-natal hours. The changes in the plasma calcium and inorganic phosphorus concentrations are suggestive of parathyroid deficiency. Response to thyrocalcitonin release and bone mineralization also play an important role in these alterations. The normal neonate exhibits the changes in the form of late rise in the urea and inorganic phosphorus concentrations which are suggestive of an impaired renal function and occurrence of tissue breakdown. The magnitude and extent of these changes in the plasma electrolytes are higher in the abnormally born infants. The infant of difficult delivery group has higher concentrations of potassium and inorganic phosphorus due to an increased degree of acidosis at birth. It undergoes a more traumatic birth, the stress of which stimulates the A.C.T.H. secretion. This endogenously released A.C.T.H. produces an altered response of the adrenocortical steroids, which is responsible for the changes seen in the plasma sodium and chloride levels. The low birth weight infant is faced with intra-uterine growth retardation which is manifested in the form of lower concentrations of minerals like magnesium and phosphorus. The degree and extent of tissue catabolism and impairment of renal function are more in the low birth weight infant. The pre-term birth is a special problem by itself. The above changes of tissue breakdown and renal function are further aggravated in the premature infant. The infant of toxæmic mother is somewhat more acidotic as compared to the

corresponding non-toxaemic neonate. The placental insufficiency in maternal toxæmia is responsible for this change which also brings about a higher magnitude of tissue breakdown thus accounting for the higher concentrations of plasma magnesium, blood urea and inorganic phosphorus observed in this group.

Synthesis of albumin by the liver during intra-uterine development of the full-term normal infant appears to be comparable to that of an adult, while it is seen to be affected by the growth retardation. Liver of the premature infant performs this function at a significantly lowered rate. A parallelism is observed in the normal placental transfer of gamma globulin in the full-term and low birth weight infants, while the peak value of the same is not attained in the premature neonate. Therefore, the concentration of gamma globulin can serve as a biochemical yardstick for the differentiation between the low birth weight and premature infant. Placental insufficiency is responsible for the low concentration of gamma globulin in the infants delivered with maternal toxæmia.